DOPPLER PARAMETERS OF HEPATIC HEMODYNAMICS IN CIRRHOTICS - ITS CLINICAL AND HISTOLOGICAL CORRELATION

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CERTIFICATE

This is to certify that the dissertation entitled **"DOPPLER PARAMETERS OF HEPATIC HEMODYNAMICS IN CIRRHOTICS – ITS CLINICAL AND HISTOLOGICAL CORRELATION"** is a bonafide work done by **Dr. KARTHIKEYAN.R** at Madras Medical College, Chennai in partial fulfilment of the university rules and regulations for award of D.M., Degree in Medical Gastroenterology (Branch-IV) under my guidance and supervision during the academic year 2011 -2014.

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DECLARATION

I solemnly declare that this dissertation entitled "**DOPPLER PARAMETERS OF HEPATIC HEMODYNAMICS IN CIRRHOTICS-ITS CLINICAL AND HISTOLOGICAL CORRELATION** was done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, during 2011-2014 under the guidance and supervision of **Prof. PUGAZHENDHI.T M.D, D.M.** This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of D.M. Degree in Medical Gastroenterology (Branch-IV).

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ABSTRACT

DOPPLER PARAMETERS OF HEPATIC HEMODYNAMICS IN CIRRHOTICS - ITS CLINICAL AND HISTOLOGICAL CORRELATION

AIM OF THE STUDY

To compare the Doppler parameters such as Hepatic artery Pulsatile and Resistive indices of compensated and decompensated cirrhotics.

To correlate these doppler parameters with clinical, biochemical and histological parameters in compensated cirrhotics and to carry out clinical and biochemical correlation in decompensated cirrhotics.

INTRODUCTION

Cirrhosis is a diffuse process characterised by fibrosis and conversion of normal liver into abnormal nodules[8].Cirrhosis is most common cause of Portal Hypertension (PHT).Cirrhosis characterised by alteration in systemic and splanchnic hemodynamics[1,2,3.] Alteration of these hemodynamics leads to Portal Hypertension. There are various methods to assess portal hypertension. Liver biopsy is the gold standard for diagnosis of liver fibrosis.

MATERIALS AND METHODS

The study was carried during the period June 2013- February 2014. 20 cases each of Compensated and Decompensated cirrhosis were involved in the study.

RESULTS

Variable	COMPENSATED	DECOMPENSATED	P value
HAPI	**1.76**	**1.92**	**< 0.001**
HARI	**0.69**	**0.87**	**< 0.001**
MELD	**9.5**	**13.8**	**< 0.001**

CONCLUSION

1. HAPI is indicator of severity of resistance due to fibrosis in both study groups.
2. HAPI and HARI similar to that of MELD are non invasive doppler parameters predicting severity of liver disease and its progression.
3. Hepatic artery pulsatile index had significant correlation with AST, indicating severe fibrosis and severe form of liver disease.
4. All patients in compensated group had cirrhosis (F4) hence histological correlation could not be carried out.

INTRODUCTION

Cirrhosis is a diffuse process characterised by fibrosis and conversion of normal liver into abnormal nodules[8].Cirrhosis is most common cause of Portal Hypertension (PHT).Cirrhosis characterised by alteration in systemic and splanchnic hemodynamics[1,2,3.] Alteration of these hemodynamics leads to Portal Hypertension. There are various methods to assess portal hypertension. Liver biopsy is the gold standard for diagnosis of liver fibrosis. Non invasive methods for assessment of PHT include Doppler Ultrasonography, hepatic vein pressure gradient, splenic pulp pressure and endoscopic variceal pressure.

Doppler Ultrasonography is inexpensive and non-invasive tool to assess focal and diffuse parenchymal changes in liver[4]. Investigators documented increase in renal resistance in cirrhotics[5,6] . Doppler parameters correlate with complications of portal hypertension. These doppler parameters used for assessment of prognosis and response to pharmacological treatment.

Hepatic artery and Renal artery Resistive index (HARI) and Pulsatile index (HAPI) were studied by **Rivolta et al**[5]. Higher the Hepatic artery Resistive indices, more is the severity of ascites and complications. Resistive index of hepatic artery is inversely related to organ perfusion.

Gaiani *et al*. (11) noted that spontaneous hepatic encephalopathy more frequent in patients with hepatofugal flow in the portal system (21% *vs.* 7.2%; $P < 0.05$).

Sacerdoti et al[7] showed that higher the RI in intrarenal arteries predicts development of Hepatorenal syndrome and poor survival. Advantages of Doppler are safe, inexpensive and painless. Disadvantages it is operator dependent. Limitations of doppler are obesity, excess bowel gas and respiratory movement.

Cirrhosis associated with increased intrahepatic resistance. It leads to increased resistance in main portal vein. **Wachsberg *et al*. (9)** pointed out that the prevalence of hepatofugal flow varied between 3% and 23%. Child and Turcotte first introduced their scoring system in 1964 which was subsequently revised by Pugh in 1973(10). Child Turcotte Pugh (CTP) score is widely used for prognosis and for clinical correlation.

There are few studies correlating Doppler parameters with clinical, biochemical and histological parameters. This study is carried to validate the correlation and to assess the outcome.

AIM OF THE STUDY

1. To compare the Doppler parameters such as Hepatic artery Pulsatile and Resistive indices of compensated and decompensated cirrhotics.

2. To correlate these doppler parameters with clinical, biochemical and histological parameters in compensated cirrhotics and to carry out clinical and biochemical correlation in decompensated cirrhotics.

REVIEW OF LITERATURE

LIVER FIBROSIS

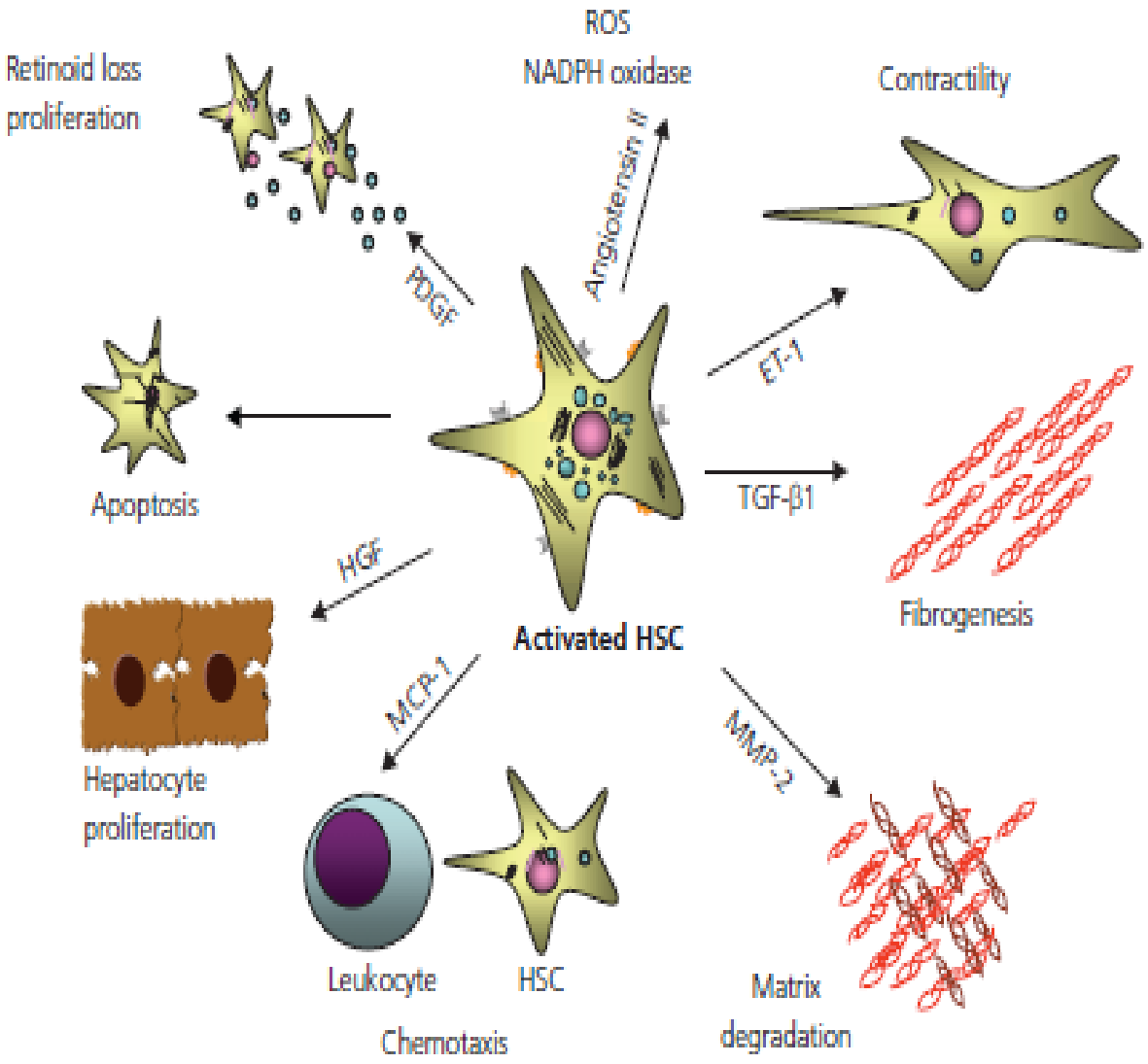
Liver fibrosis is accumulation of extracellular matrix proteins (ECM) in chronic liver diseases[12]. Most common causes of liver fibrosis are Chronic Viral Hepatitis, Alcohol abuse and Non alcoholic steatohepatitis. Accumulation of ECM proteins leads to scar and nodule formation leads to cirrhosis. Cirrhosis leads to increased intrahepatic resistance, hepatic insufficiency and portal hypertension.

Most important cell involved in Hepatic fibrosis is Hepatic stellate cell (HSC) or "Ito cell". Normally it is involved with vitamin A storage[14]. First described by Von Kuppfer in 1876. Onset of fibrosis is insidious progresses to cirrhosis in 15-20 yrs.

Liver fibrosis is influenced by genetic and environmental factors. Chronic Viral hepatitis and Alcohol account for half of cases[1].

Major clinical complications of cirrhosis include ascites, renal failure, hepatic encephalopathy, and variceal bleeding. Patients with compensated cirrhosis remain free of complications compared to decompensated cirrhotics.

Properties of activated stellate cell



CAUSES OF LIVER FIBROSIS

Chronic viral diseases	Autoimmune diseases
Hepatitis B	Autoimmune hepatitis
Hepatitis C	Primary Biliary Cirrhosis
Hepatitis D	Primary sclerosing cholangitis
	Graft versus host disease

Drugs	Metabolic causes
Methotrexate	Nonalcoholic steatohepatitis
α methyl dopa	Wilsons disease
Amiodarone	Hemochromatosis
Vitamin A intoxication	α1 Antitrypsin deficiency
	Type 4 Glycogen storage disease
	Tyrosinemia

Vascular

Budd chiari syndrome

Venoocclusive disease

IVC thrombosis

Rt.sided Heart failure.

Miscellaneous

Secondary biliary cirrhosis

Cryptogenic

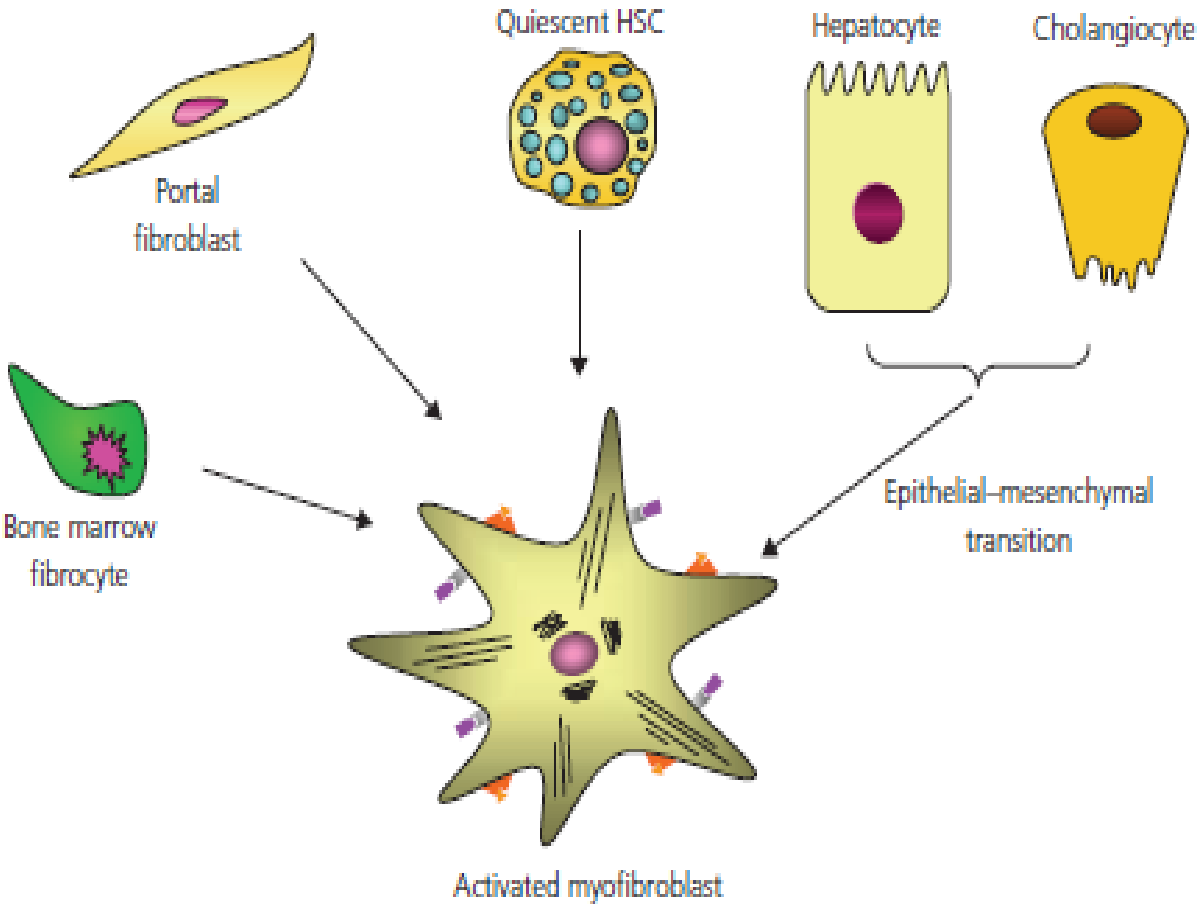
Cellular mechanisms of liver fibrosis

HSC activation is the key step in hepatic fibrogenesis. These cells contribute 15% of liver population. Hepatocytes and Kuppfer cells promote HSC activation by lipid peroxides leads to oxidative stress. Antioxidants attenuate liver fibrogenesis by inhibiting these cells. Most important growth factor involved is PDGF.

Interaction of HSC with extracellular matrix important in profibrogenic behavior. These cells play active role in hepatic inflammation[15.] Myofibroblasts are other cells known to have fibrogenic potential.

Sources of Myofibroblasts

Various sources of Myofibroblasts are Hepatocyte, Cholangiocyte, portal fibroblast, Quiescent HSC.



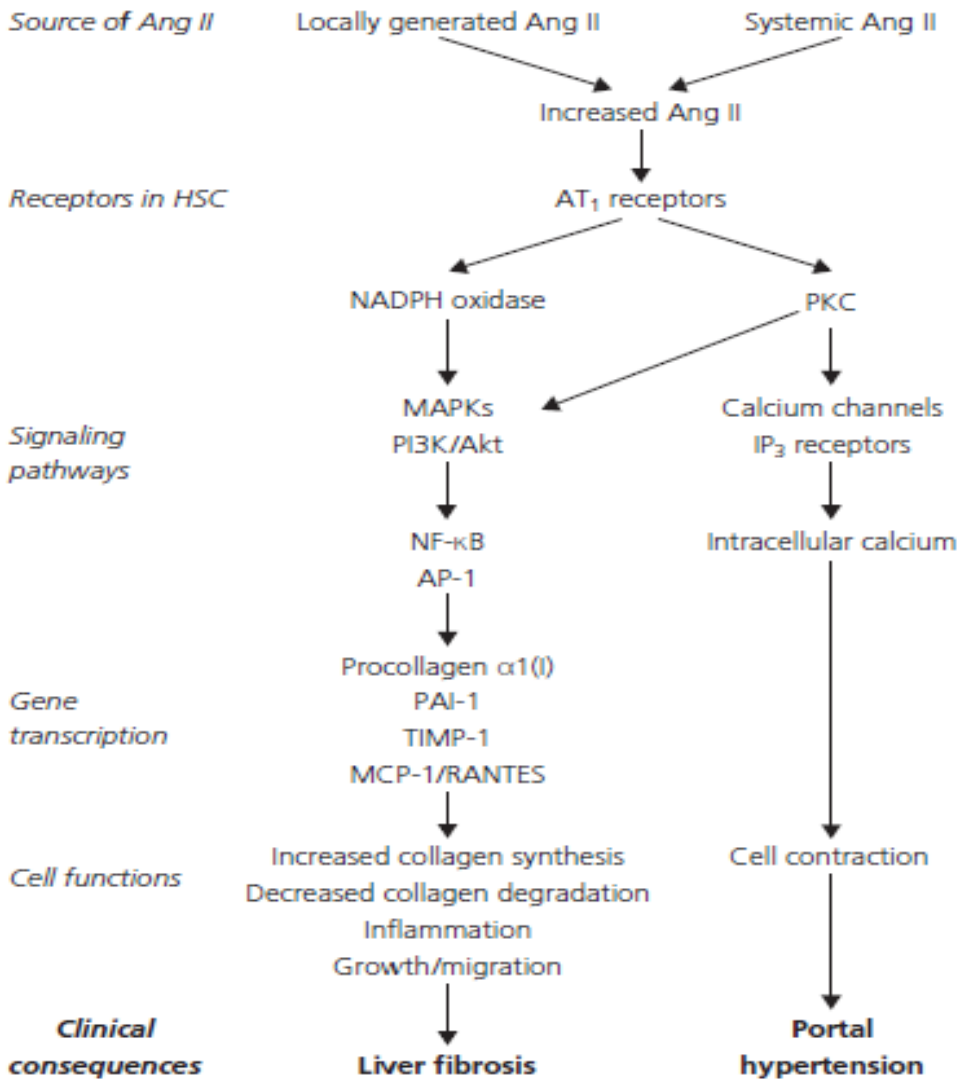
MOLECULAR MECHANISMS IN LIVER FIBROSIS

Liver fibrosis is due to alteration and change of composition of Extracellular matrix proteins[16]. Major extracellular matrix component in fibrotic liver is Type 1 collagen. Mechanisms of liver fibrosis include oxidative stress, hypoxia and angiogenesis. Oxidative stress play major role in hepatic fibrogenesis. NADPH complex plays novel regulator in liver fibrogenesis.

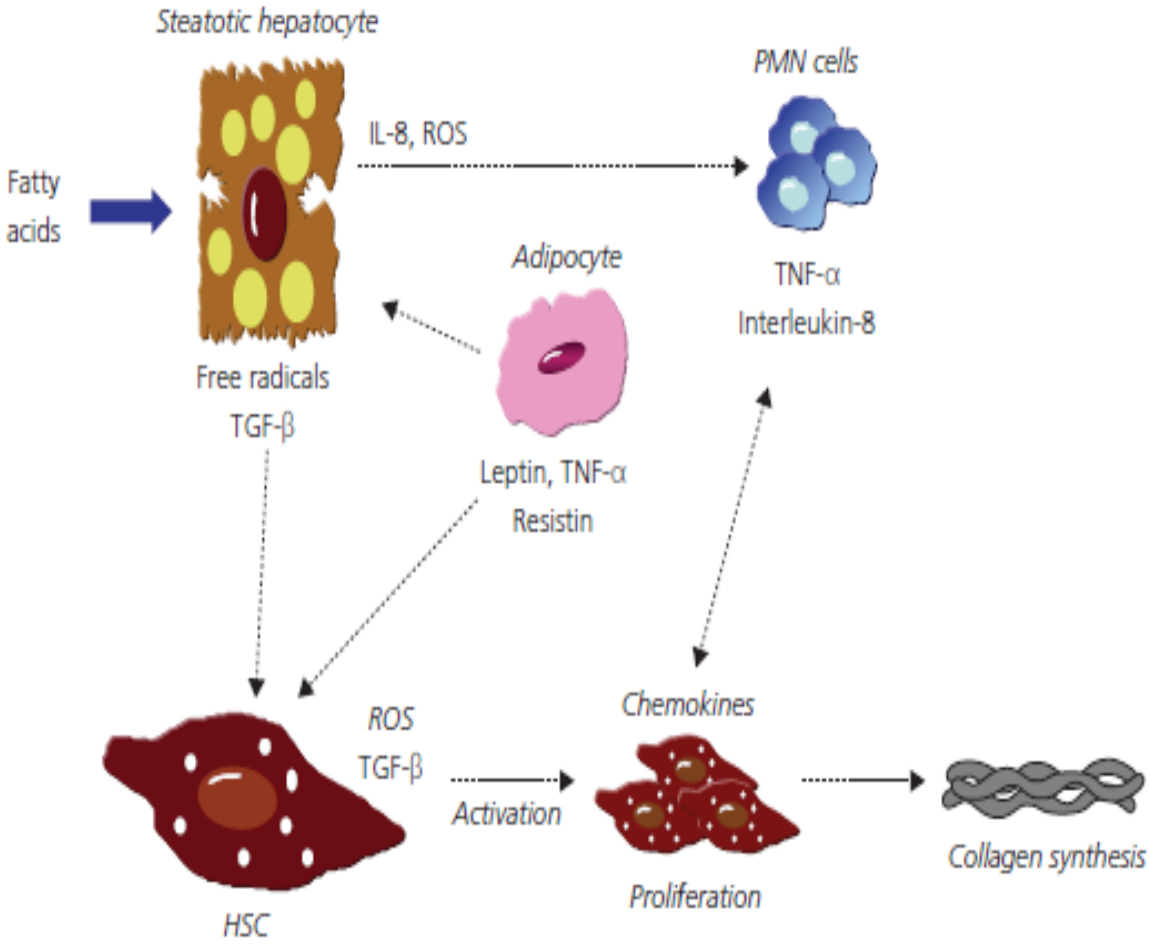
Several cytokines regulate liver fibrogenesis. TGF-β is most effective fibrogenic cytokine in liver. Several growth factors such as PDGF, EGF and IGF play an important role of which Platelet derived growth factor (PDGF) is most important.

Angiotensin2 plays important role in hepatic fibrogenesis[17.]

PATHOGENIC EFFECT OF RENIN ANGIOTENSIN SYSTEM

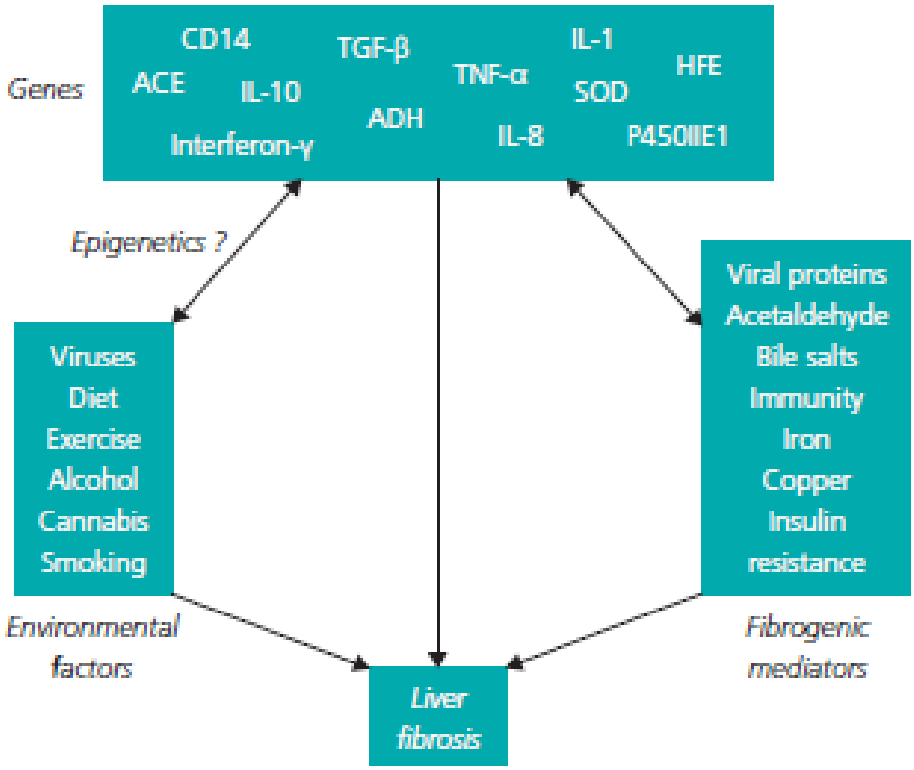


PATHOGENESIS OF LIVER FIBROSIS



GENETIC DETERMINANTS OF LIVER FIBROSIS

Liver fibrosis is a condition in which multiple genes interact with environmental factors[18.] ·Alcoholic liver disease characterized by interaction by genetic, environmental and behavioral factors. Studies have identified polymorphisms in a number of candidate genes that may influence the progression of liver fibrosis in humans[19..]



PORTAL HYPERTENSION (PHT)

It is a pathologic increase in hydrostatic pressure in the portal venous system. Ninety percent of patients with PHT have sinusoidal or post sinusoidal portal hypertension. It is caused mainly by cirrhosis due to Hepatitis B or C infection.

Cirrhosis characterised by fibrosis and formation of nodules. Formation of nodules leads to distortion of hepatocyte architecture and leads to portal hypertension. Normal portal pressure is 5- 10 mmHg. Normal Portal vein (PV) flow is Hepatopetal and usually monophasic with some fluctuation due to respiration and cardiac activity. Normal Portal vein diameter is 13 mm.

Dilatation of portal vein is when more than 13mm occurs in 56% of pts. Normal caliber of PV does not however exclude PHT. Upper limit of normal splenic and superior mesenteric vein are 10-12 mm. Splenomegaly usually associated with dilatation of Splenic vein. PHT is measured by several methods:

1. Doppler ultrasonography
2. Hepatic vein pressure gradient
3. Splenic pulp pressure
4. Portal vein pressure
5. Endoscopic variceal pressure

Hepatic vein pressure gradient (HVPG) is wedge hepatic pressure- free hepatic vein pressure. First described in 1951.

Indications for measurement of HVPG

1. To monitor portal pressure
2. Prognostic marker
3. Assess risk of hepatic resection in patients with cirrhosis
4. Delineate cause of portal hypertension
5. Endpoint in trials using drugs for portal hypertension

DOPPLER FINDINGS IN PHT

1. Dilated splenoportal axis

2. Portosytemic collaterals

3. Hepatofugal flow

4. Phasic variation of Hepatic vein reduced or absent

5. SMA shows decrease pulsatility index.

In cirrhosis the portal vein blood flow is reduced. It is compensated by hepatic arterial flow. In setting of cirrhosis measurement of Hepatic arterial Pulsatile index and Resistive indices are markers of resistance. Hence measurement of these parameters is more valid than measurement of routine doppler parameters.

PULSATILITY AND RESISTIVE INDICES

Hepatic artery resistance can be measured by measuring the Resistive index (RI) and Pulsatile index (PI). Although these indices are closely correlated, hepatic artery Pulsatile index is considered a better index of resistance. Since these indices are ratios which can be measured by calculating the peak systolic velocity, peak diastolic velocity and mean velocity they are less likely to be affected by subjective measurements.

Hepatic arterial resistive indices have been shown to increase in cirrhosis and portal hypertension. **Schneider et al [43]** reported that hepatic arterial Pulsatile index is higher in cirrhotics compared to controls in his study and found direct correlation with portal pressure. Hepatic arterial pulsatility index correlated with non-invasive assessment of portal hypertension.

The pathological mechanisms resulting in PHT are narrowing of vascular space by fibrous tissue, compression by regenerative nodules, increased contractility in response to vasoconstrictors.

OTHER DOPPLER INDICES IN VARIOUS STUDIES

1. **Portal vein congestive index (PVCI)** is defined as ratio of cross sectional area of the extra-hepatic portal vein to time averaged mean velocity of blood flow in the portal vein. Elevated in cirrhotics.
2. **Doppler perfusion index (DPI)** is the ratio of hepatic arterial blood flow (normal below 20 %) to the total liver blood flow (hepatic arterial and portal venous blood flow). Elevated in Cirrhotics.

   Doppler Perfusion Index(DPI) = Blood flow of hepatic artery

   (Blood flow of hepatic artery + blood flow of portal vein)

   DPI= BFHA / (BFHA+BFPV)

Gaiani *et al* [20] showed that 80.4% of cirrhosis can be detected in patients with compensated liver diseases of various etiologies using a US scoring system based on two US parameters..

Colli *et al* [21] reported that US can detect severe fibrosis or cirrhosis with a specificity of 0.95 and a sensitivity of only 0.54.

Zheng *et al* [22] studied value of US in evaluation of liver fibrosis and compensated cirrhosis in comparison with serology and histology, found that hepatic parenchymal echo pattern, liver surface and thickness of gallbladder wall are three independent predictors of liver fibrosis.

Diagnostic accuracy of USG for compensated cirrhosis is 80.5%.

Doppler USG measurement does not represent HVPG.

Schalm et al [23] showed that even if histology shows no evidence of cirrhosis but if there is fibrosis and architectural distortion, diagnosis of cirrhosis should be made if there is ultrasound evidence of cirrhosis.

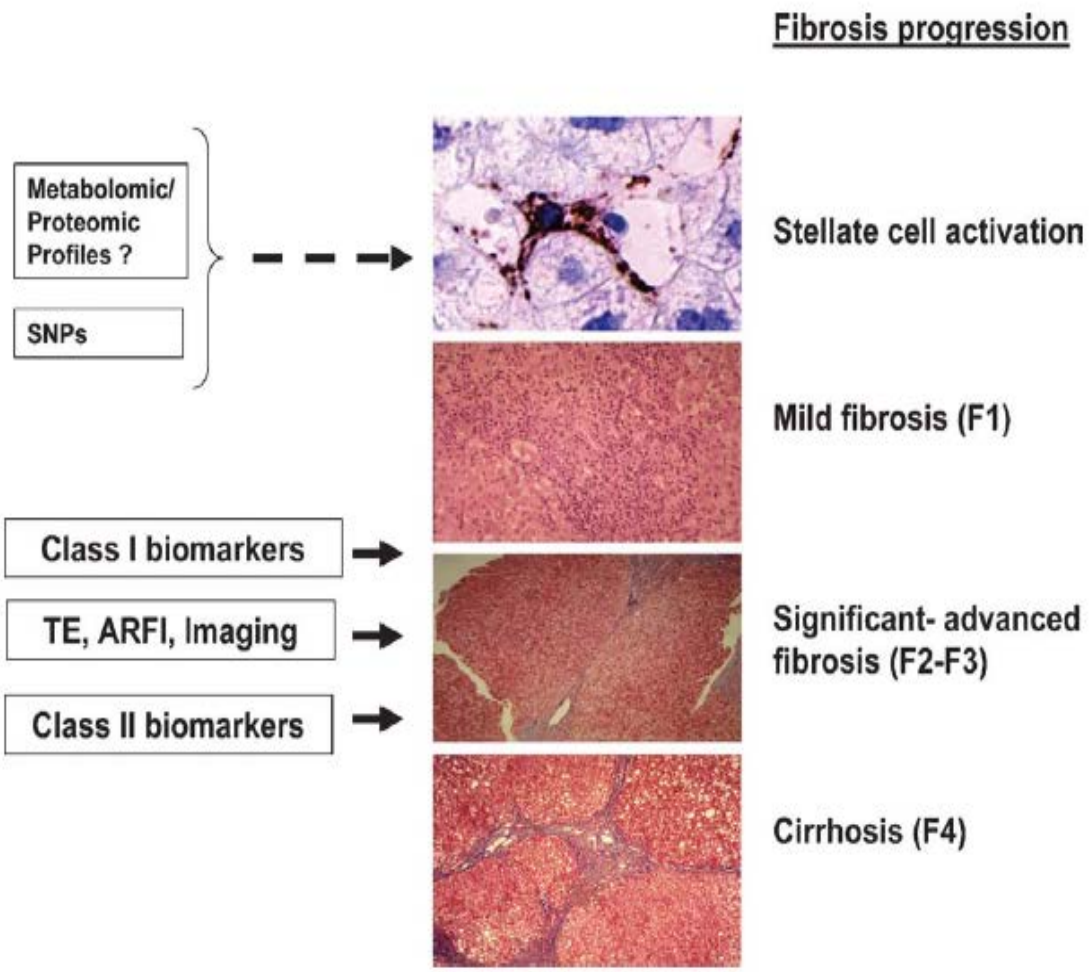
Hemodynamics in hepatic blood flow influenced by extent of fibrosis, chronic inflammation, presence and size of varices and portosystemic shunts.

Other noninvasive method of assessing liver fibrosis is Transcient elastography.

TRANSCIENT ELASTOGRAPHY (FIBROSCAN)

Mild amplitude and low frequency wave is transmitted to tissue producing shear wave within liver. It is noninvasive useful in several liver diseases. Limitations not used in morbid obesity. Liver stiffness always is not a surrogate marker of fibrosis. Useful in mild and advanced fibrosis not useful in intermediate fibrosis.

NON INVASIVE ASSESSMENT OF FIBROSIS



Hepatic artery Pulsatile index (HAPI) and Resistive indices (HARI) calculated using following formulas:

HEPATIC ARTERY

RESISTIVE INDEX (RI) = Peak Systolic velocity -- Peak Diastolic velocity

Peak systolic velocity

HEPATIC ARTERY

PULSATILE INDEX (PI) = Peak Systolic velocity -- Peak Diastolic velocity

Mean velocity

Hepatic artery Pulsatile and Resistive indices were studied by assessing blood flow velocity waveform analysis by doppler studies.

Hyperdynamic circulation in splanchnic and systemic areas seen in advanced liver diseases. **Schrier et al** suggested that these hemodynamic changes can be seen in early stages of cirrhosis.

Portal Doppler was done. In it various parameters such as assessment of portal vein size, liver echoes, presence of splenomegaly, and presence of collaterals were assessed. Normal individual's diameter of portal vein should not exceed 13 mm in supine posture and quiet respiration.

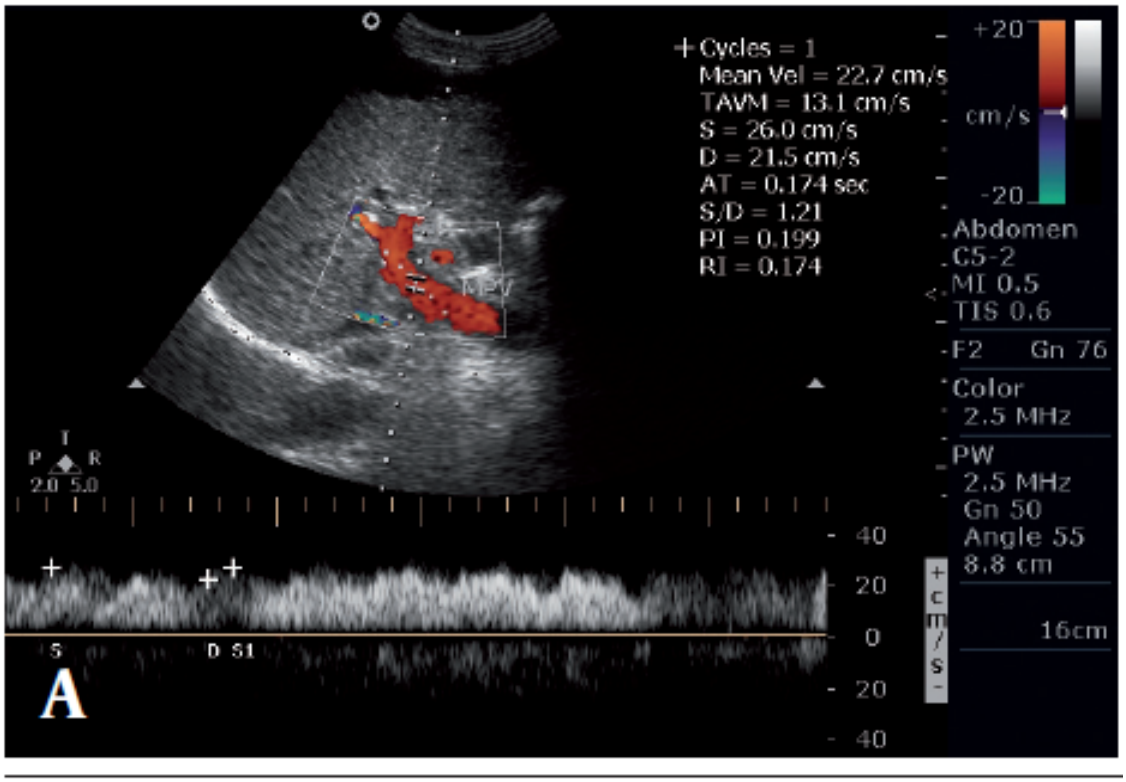
PHT defined when size of PV more than 13mm. It has high specificity of 100% and sensitivity of 45- 50% [26, 27.] In normal individuals the diameter of portal vein increases by 70 -100% from quiet respiration to deep inspiration.

In normal individuals flow is Hepatopetal throughout entire cardiac cycle. Flow velocity is about 15- 18 cm/sec. The following changes seen with portal hypertension:

1. Flow becomes continuous
2. Bidirectional
3. Hepatofugal
4. Development of collaterals

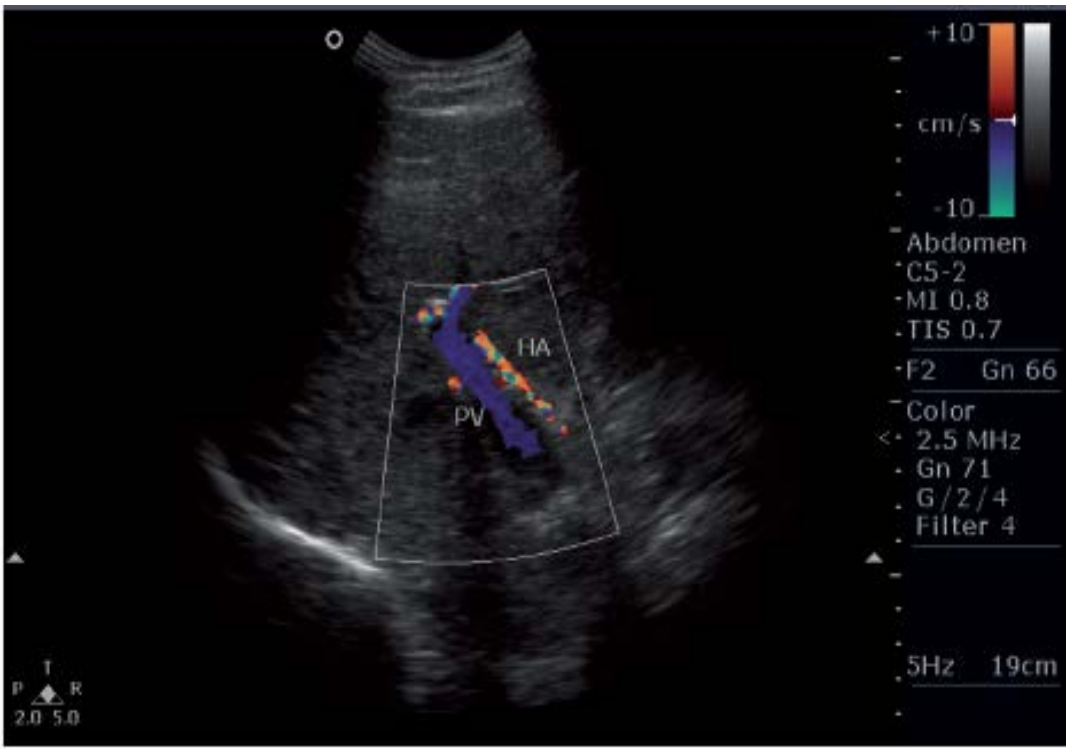
Cirrhosis is characterized by increased intrahepatic resistance. This leads to resistance to hepatopetal flow in main portal vein. This leads to opening of collaterals.

HEPATOPETAL FLOW



With progressive intrahepatic resistance leads to reversal of flow in main portal vein leads to hepatofugal flow. Wachsberg et al [28] reported prevalence of Hepatofugal flow in 3- 23% in his studies.

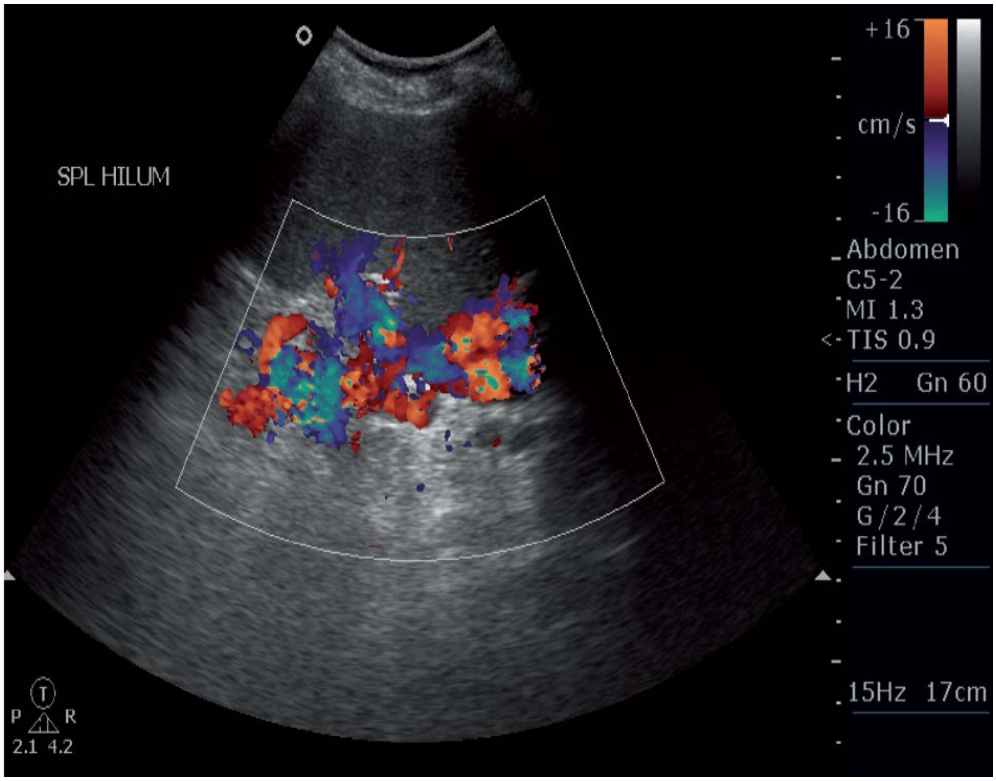
HEPATOFUGAL FLOW



Taourel et al [29] reported no correlation between degree of PHT and development of esophageal varices. **Ohnishi et al [30]** reported decreased portal velocity and hepatofugal flow associated with development of splenic varices.

Gaiani et al [31] reported spontaneous hepatic encephalopathy incidence high in patients with Hepatofugal flow.

SPLENIC VARICES



LIVER BIOPSY

Liver biopsy is the gold standard for diagnosis of liver fibrosis. The tissue obtained represents 1/ 50,000 th of liver tissue. Ideal biopsy sample should contain at least 11 portal tracts. There are several methods of liver biopsy

1. Percutaneous route
2. Transjugular route
3. Transthoracic route

Role of liver biopsy:

1. For diagnosis
2. For assessment of prognosis
3. For assisting in management of therapeutic decisions.

INDICATIONS

1. Unexplained elevation of liver enzymes.
2. Elevated liver enzymes for 6 months or longer.
3. Assessment of response to therapy of chronic liver disease
4. Multiple parenchymal liver diseases.
5. Fever of unknown origin.
6. Focal or diffuse abnormalities on imaging.
7. Staging / Prognosis of liver diseases.

PREBIOPSY WORK UP

1. Prothrombin time/ INR and partial thromboplastin time should be assessed.
2. Platelet count
3. Hemoglobin
4. Blood urea nitrogen and creatinine
5. Bleeding time
6. Antiplatelet/ warfarin / heparin to be stopped.

CONTRAINDICATIONS

1. Uncooperative patient.
2. Impaired coagulation
3. Severe uncorrected anemia
4. Significant ascites
5. Skin infection at needle insertion site.
6. High grade extrahepatic Biliary obstruction
7. Cholangitis / Uremia
8. Leukemia/ Myelofibrosis
9. Echinococcal cysts

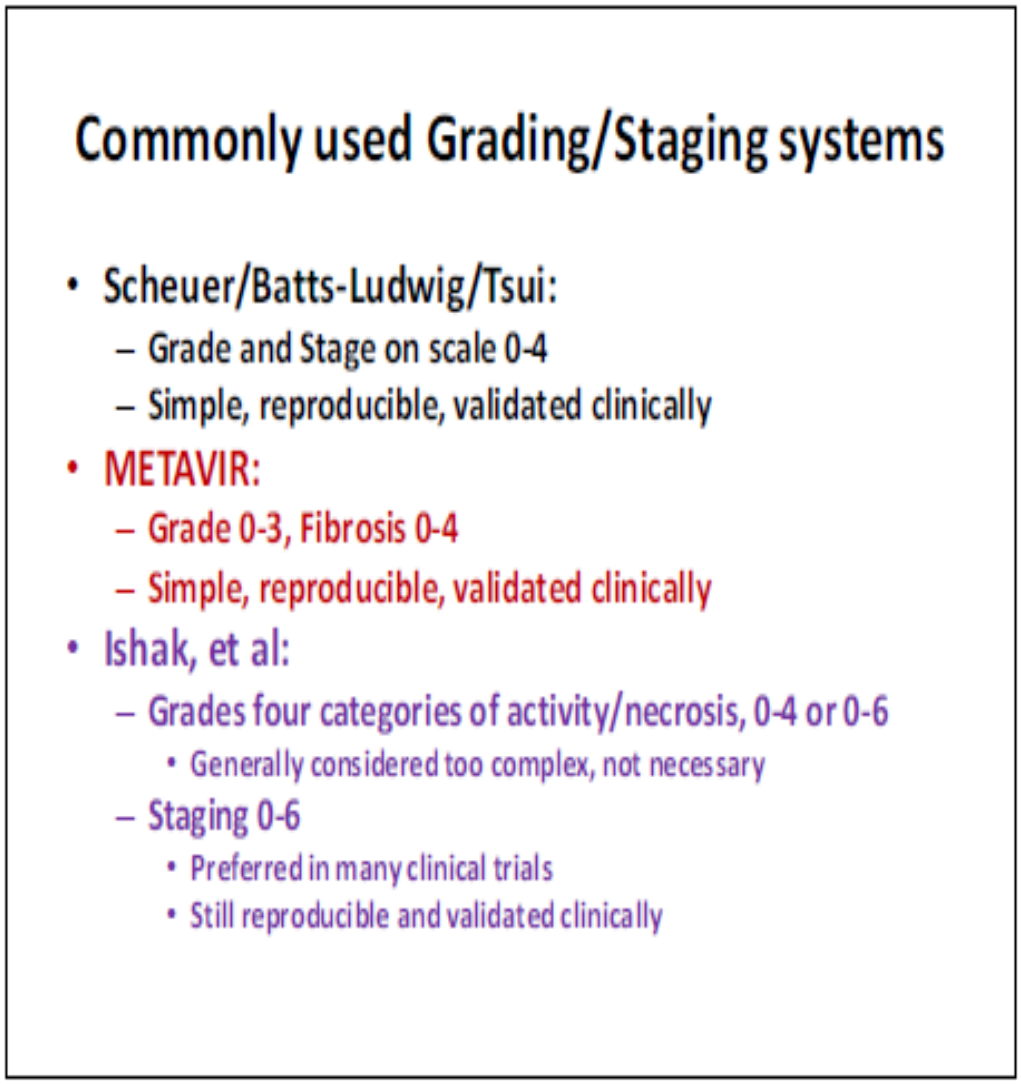
BIOPSYGUN

COMPLICATIONS

1. Pain is the most common complication seen in 3-33% pts
2. Bleeding
3. Hypotension
4. Hemothorax
5. Hemoperitoneum
6. Hematoma
7. Hemobilia
8. Gall bladder perforation

Most complications occur within 2hrs. Patient to be monitored every 15 minutes for 1hr, thereafter every 30 minutes for 2 hours.

FIBROSIS SCORING SYSTEMS



METAVIR AND LUDWIG SCORES

METAVIR

2-letter, 2-number system similar to Scheuer
Used extensively in France

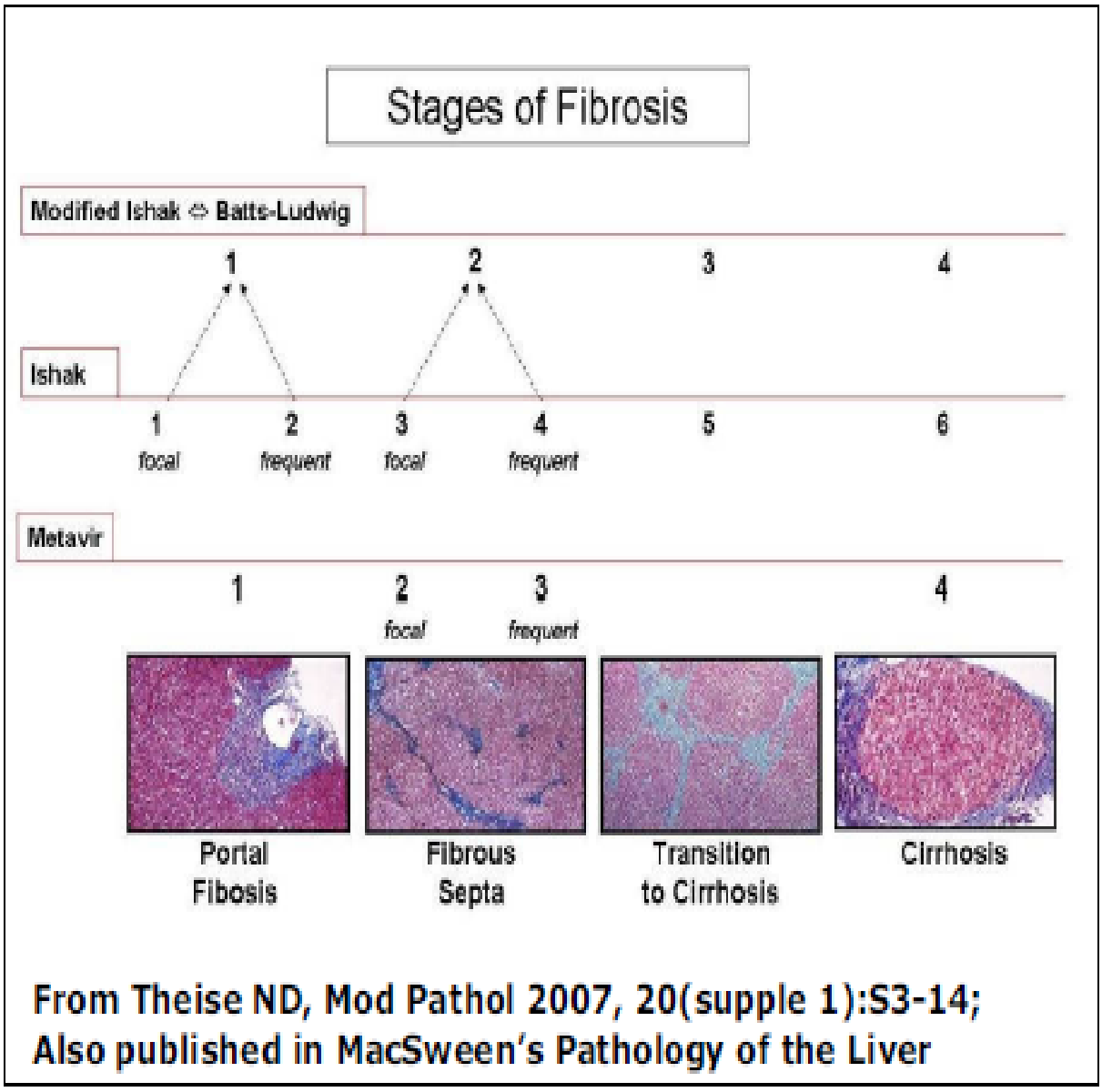
F = fibrosis

- F0 = no fibrosis
- F1 = portal fibrosis without septa
- F2 = portal fibrosis with rare septa
- F3 = numerous septa, not cirrhosis
- F4 = cirrhosis

Scheuer/Batts,Ludwig/Tsui
Fibrosis scoring for Chronic Hepatitis

Stage	Description
0	No fibrosis, normal amount of connective tissue
1	Portal/periportal fibrosis
2	Septal fibrosis
3	Bridging fibrosis with architectural distortion.
4	Cirrhosis, probable cirrhosis

Comparison of Fibrosis Scores



MATERIALS AND METHODS

This is a cross sectional study which was done in Department of Medical Gastroenterology in Rajiv Gandhi Government General Hospital Chennai. The study was carried during the period June 2013- February 2014. 20 cases each of Compensated and Decompensated cirrhosis were involved in the study.

INCLUSION CRITERIA

1. Newly diagnosed cases of compensated cirrhosis were selected for study based on USG imaging showing nodular appearance of liver, spleen more than 12 cm and presence of esophageal varices > 2 on endoscopy based on AASLD guidelines.
2. Cases of Decompensated cirrhosis with ascites, jaundice and encephalopathy were selected.

EXCLUSION CRITERIA

1. Patients with co morbid conditions such Intrinsic Renal disease, Diabetes and Hypertension were excluded.
2. Patients with serum creatinine more than 1.4 mg.
3. Patients with malignancy

DATA COLLECTION

20 cases of newly diagnosed Compensated cirrhosis and 20 cases of Decompensated cirrhosis were selected based on inclusion criteria. Various parameters such as age, sex anthropometry were obtained. Clinical features such as presence of ascites, jaundice, hepatic encephalopathy, UGI bleed were noted by interviewing and thorough examination of the patients. Clinical features suggestive of chronic liver disease found during examination were recorded. Biochemical parameters such as complete blood count, blood sugar, urea, creatinine, electrolytes, liver function tests, prothrombin time were collected. Imaging such as ultrasound abdomen with Doppler study, CT scan, was done as per the requirements. Liver biopsy was done in patients for diagnosis and grading of fibrosis. CTP and MELD scores calculated in these patients based on these parameters.

GRADING OF VARICES

These pts were subjected to upper gastrointestinal endoscopy for assessment of presence or absence of esophageal varices was made. **Pacque**t **classification** was adopted for grading of esophageal varies.

Grade 0	No varices
Grade I	Varices, disappearing with insufflation
Grade II	Larger, clearly visible, usually straight varices, not disappearing with insufflation
Grade III	More prominent varices, locally coil-shaped and partly occupying the lumen
Grade IV	Tortuous, sometimes grape-like varices occupying the esophageal lumen

Pacquet classification for grading of esophageal varices.

PORTAL DOPPLER

Doppler studies were performed after overnight fasting. Doppler study was performed with Duplex Doppler apparatus with color Doppler sonographer and 3.75 MHz convex transducer is chosen for assessment of deep vessels. The Hepatic artery and portal veins best interrogated by Doppler ultrasound of porta hepatis using oblique intercostal scans. Parameters such as Hepatic artery Peak systolic velocity and Peak diastolic velocity calculated .Using these parameters Hepatic artery Pulsatile index (HAPI) and Resistive index (HARI) were calculated as follows:

HEPATIC ARTERY

RESISTIVE INDEX (RI) = $\frac{\text{Peak Systolic velocity -- Peak Diastolic velocity}}{\text{Peak systolic velocity}}$

HEPATIC ARTERY

PULSATILE INDEX (PI) = $\frac{\text{Peak Systolic velocity -- Peak Diastolic velocity}}{\text{Mean velocity}}$

MELD score was calculated in these 2 populations.

MELD is calculated using the following log formula

0.957 X loge (creatinine mg/dl) + 0.378 X loge (total bilirubin [mg/dl]) +1.120 X loge (INR) + 0.643.

Limitations of MELD

1. Inter laboratory variations measuring creatinine.
2. Gender differences in creatinine
3. INR variations.

Criteria	1	2	3
Encephalopathy	None	Grade 1 or 2	Grade 3 or 4
Ascites	None	Mild to moderate	Large or refractory to diuretics
Bilirubin (mg/dl)	<2	2-3	>3
Albumin	>3.5	2.8-3.5	<2.8
Prothrombin time (Seconds prolonged)	<4	4-6	>6
Class A 5-6 points, Class B 7-9 points, Class C 10-15 points			

CHILD TURCOTTE PUGH SCORE

CTP score is calculated based on ascites, hepatic encephalopathy, and various other laboratory parameters such as bilirubin, albumin and prothrombin time. Various grades such as CTP A, B, and C were given. It is used as a prognostic marker. Child Pugh score was assessed.

LIVER BIOPSY

Liver biopsy carried out in cases of compensated cirrhosis after assessment of coagulation profile such as prothrombin time, INR, bleeding time and clotting time. Platelet count to be assessed. Antiplatelet drugs to be stopped 10 days prior, warfarin to be stopped 5 days prior, heparin to be stopped 12-24 hours prior to biopsy.

Procedure

1. Procedure is explained to the patients and consent is obtained.
2. Test dose of 1% lignocaine is given and tested for anaphylaxis.
3. Patient is put in reclined posture, head end elevated with arms raised above the head.
4. Maximum point of liver dullness is made out by percussion and is marked. Biopsy is done in Rt.9^{th} ICS in midaxillary line.
5. Area is sterilized on scrubbing with betadine solution.
6. Patient is explained to hold the breath at end of expiration, so that the liver does not move with respiration
7. 5ml of lignocaine is infiltrated into skin.
8. Using liver biopsy gun, biopsy is done with “firing” technique.

Biopsy specimen fixed in 10% neutral formalin and stained with Hematoxylin and eosin or Masson trichrome stains. Ideal biopsy specimen should be at least 2 cm in length and not in fragments. It should contain greater than 11 portal tracts .Liver biopsy to be carried with platelet transfusion when platelet count is < 50,000. Patient is observed every 15 minutes for 1 hour and every 30 minutes for 2 hours. Most common complication seen in our patients was pain at biopsy site which was treated with analgesics.

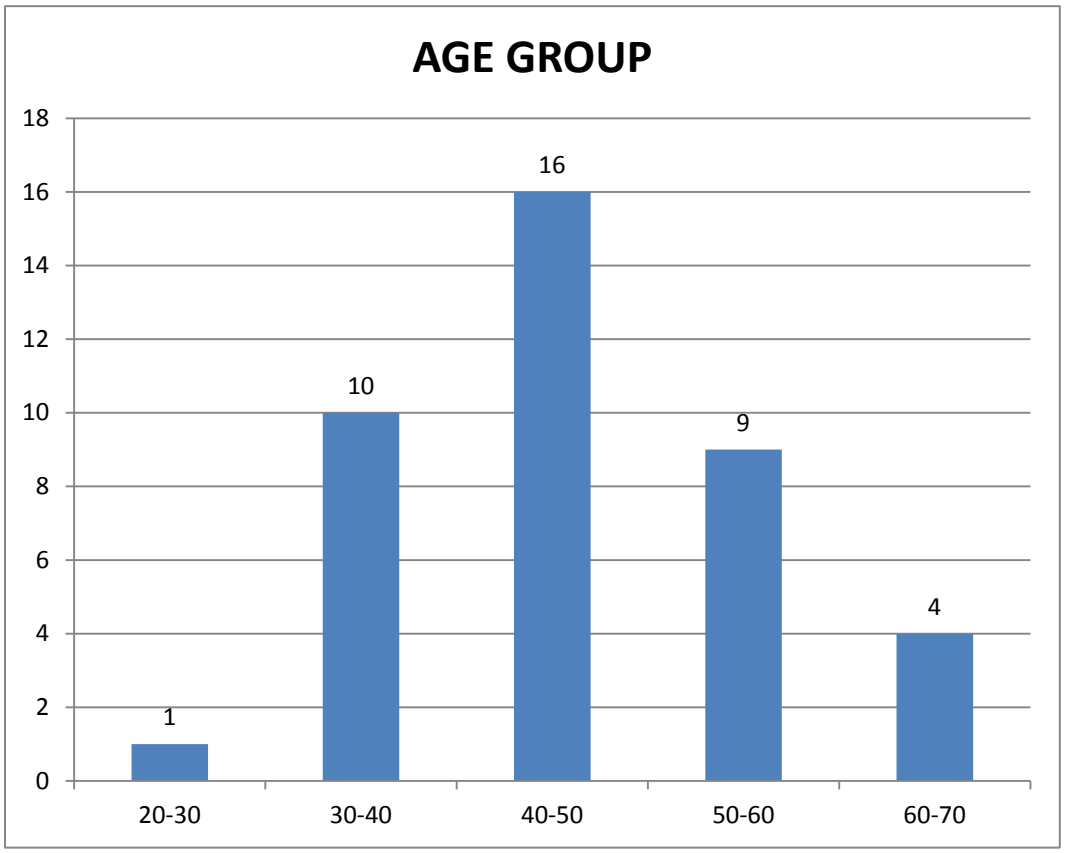
SAMPLE LIVER BIOPSY SPECIMEN

Statistical analysis

All the data were entered on a excel sheet. Mean and median were calculated for appropriate variables. Variables significant by univariate analysis were again compared by multivariate analysis. Pearson correlation coefficient and paired t test used for analysis. P value less than 0.05 was taken as significant. P value less than 0.001 was take to be very statistically significant. Statistical analysis was done by SPSS 16 software.

RESULTS

In our cross sectional study a total of 40 pts with chronic liver disease were enrolled. Out of which 20 cases each of compensated and decompensated cirrhosis were selected. There were 32 males and 8 females. The mean age of presentation was 45.5years in compensated group and 48.4 years in decompensated group. Most common age group was between 40-50 years which included 16 patients.



SEX DISTRIBUTION

Majority of the study population were males

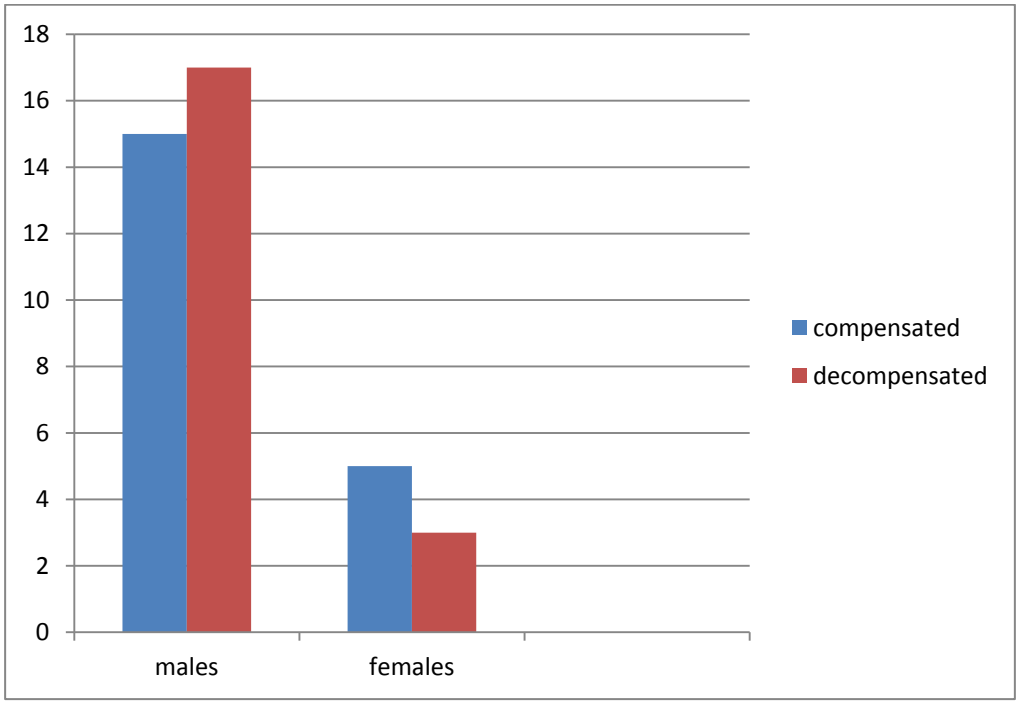


Figure 1

Out of 40, history of alcohol consumption present in 30 cases. History of upper gastrointestinal bleed present in 28 cases. Encephalopathy present in 9 cases seen in decompensated group. Splenomegaly present in 34 cases. Mean hemoglobin was 11gm% in compensated and 8.5gm% in decompensated group.

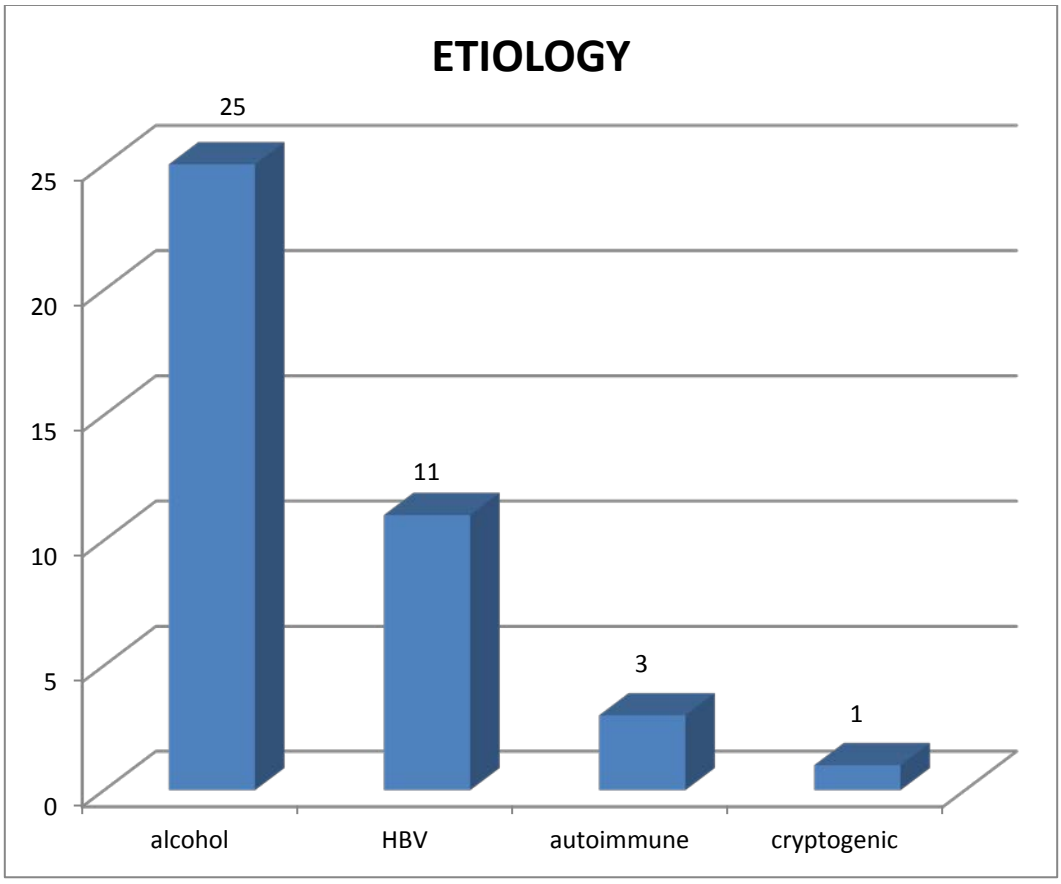


Figure 2

Etiological evaluation done alcohol found to be the common etiological factor followed by Hepatitis B viral infection and autoimmune hepatitis.

Ascites was present in 20 out of 40 cases. Grading of ascites done as a component of Child Pugh score

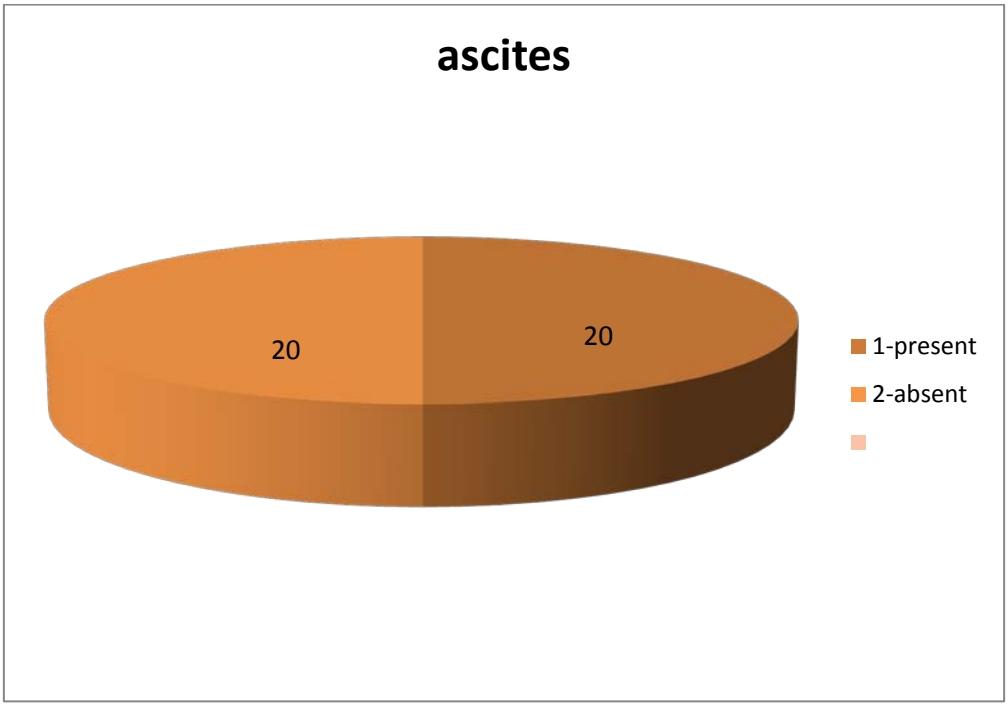


Figure 3

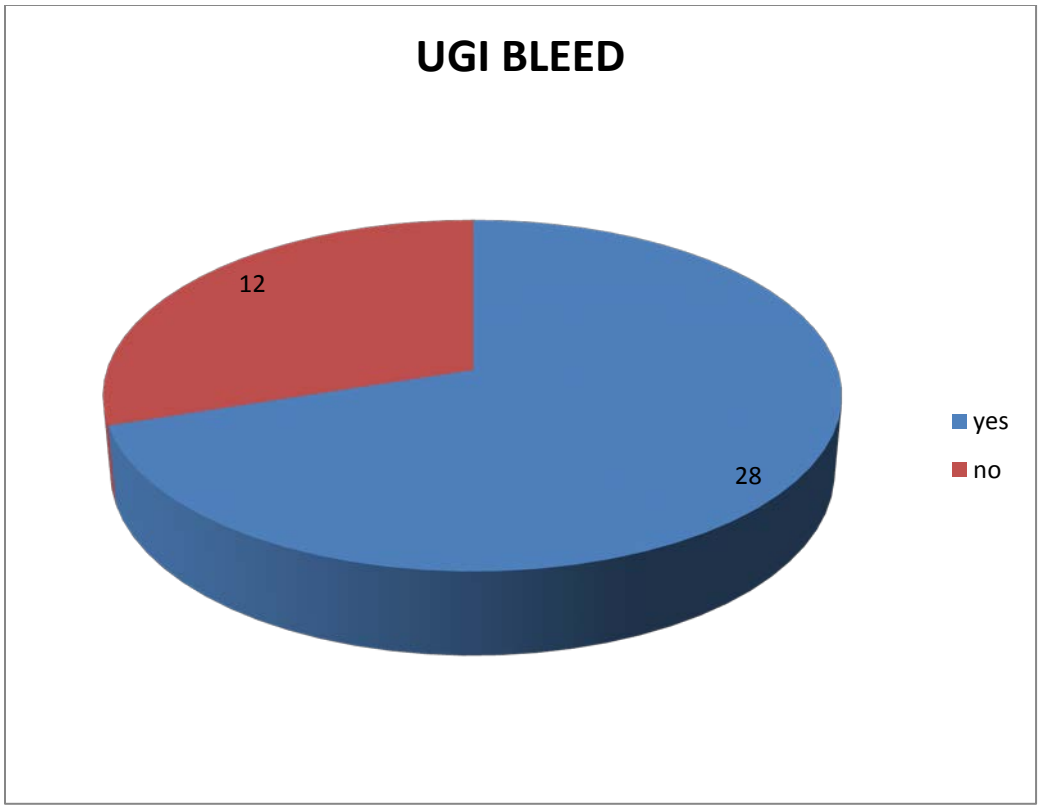


Figure 4

History of UGI bleed present in 28 cases in both groups. The mean hemoglobin was low in both groups.

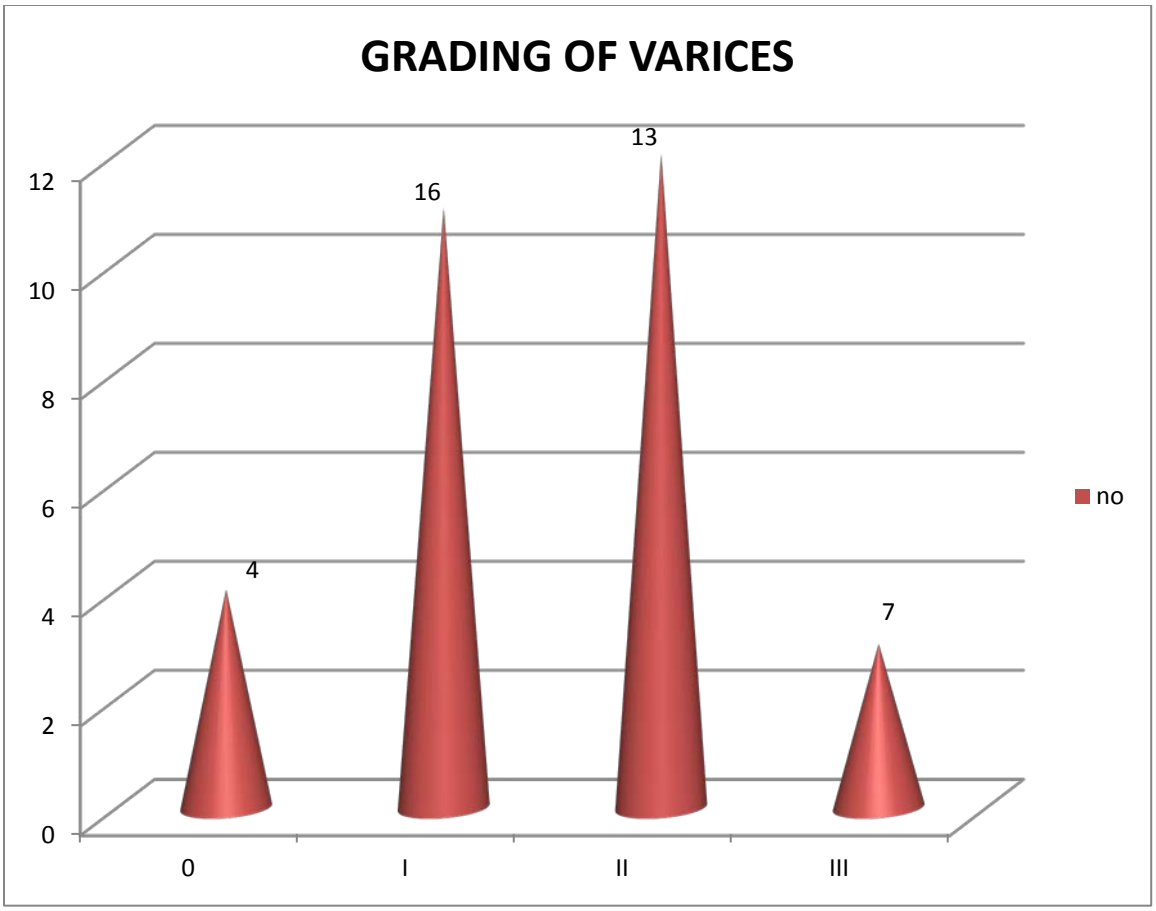


Figure 5

Grading of varices done by Pacquet classification. No varices found in 4 cases. Grade 1 varices in 16 cases. Grade 2 in 13 and Grade 3 in 7 cases.

Average platelet count in compensated group was 84,600 and 58,700 in decompensated group. The albumin level in compensated group was 3.665gm and 3.25 gm in decompensated. The average bilirubin in compensated was 1.118 mg% and 7.145 mg% in decompensated group. The prothrombin time in compensated was 17.4 and 19.5seconds in decompensated group. AST in compensated group was 63 IU and 159 IU in decompensated group.

CTP score was calculated based on presence and severity of ascites, grade of encephalopathy, bilirubin, albumin and prolongation of prothrombin time. Compensated cirrhosis were in CTP A, whereas decompensated 10 were in CTP B and 10 were in CTP C. MELD score was calculated. 18 had MELD score < 10, 22 had MELD score > 10.

Various Doppler parameters such as portal vein diameter, phasic variation, and direction of flow and presence of collaterals were assessed. Most important parameters of resistance such as Hepatic artery Peak systolic flow velocity and peak diastolic velocity were calculated. From these values, pulsatile index and Resistive indices of Hepatic artery was calculated.

Liver biopsy was carried out after prebiopsy workup. Biopsy was done in cases of compensated cirrhosis. Using Metavir fibrosis score degree of fibrosis calculated. Since study population selected was compensated all cases had F4 cirrhosis.

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Table Showing Percentage of Splenomegaly

			Group		
			Compensated	Decompensated	Total
Splenomegaly	Present	Count	17	17	34
		% within Splenomegaly	50.0%	50.0%	100.0%
		% within Group	85.0%	85.0%	85.0%
	Absent	Count	3	3	6
		% within Splenomegaly	50.0%	50.0%	100.0%
		% within Group	15.0%	15.0%	15.0%
Total		Count	20	20	40
		% within Splenomegaly	50.0%	50.0%	100.0%
		% within Group	100.0%	100.0%	100.0%

Table shows presence of Splenomegaly in 34 cases and its absence in 6 cases.

Table Showing Comparison of HA-PI with AST in both groups

GROUP	HAPI	AST(IU)	
COMPENSATED	1.75	63	P value < 0.05
DECOMPENSATED	1.92	159	

The above table shows comparison of HAPI with AST. The mean AST in compensated group is 63 and 159 in decompensated group.HAPI is associated with AST with significant p value of < 0.05.

Analysis of laboratory values

	Group	N	Mean	Std. Deviation
WBC	Compensated	20	6045.00	1284.513
	Decompensated	20	6220.00	1616.233
HB	Compensated	20	11.065	1.7748
	Decompensated	20	8.565	.9713
Platelets	Compensated	20	84600.00	21724.107
	Decompensated	20	58700.00	10367.459
RBS	Compensated	20	125.10	11.747
	Decompensated	20	129.00	18.607
Urea	Compensated	20	30.65	4.591
	Decompensated	20	31.15	6.620
Creatinine	Compensated	20	.980	.1240
	Decompensated	20	.975	.1713
proteins	Compensated	20	6.50	.513
	Decompensated	20	6.25	.444
albumin	Compensated	20	3.665	.3990
	Decompensated	20	3.275	.3477
AST	Compensated	20	63.65	12.840
	Decompensated	20	159.15	49.242
ALT	Compensated	20	45.60	6.793
	Decompensated	20	110.35	30.051
Bilirubin	Compensated	20	1.118	.5056
	Decompensated	20	7.145	1.7212
PT	Compensated	20	17.40	1.142
	Decompensated	20	19.59	2.334
INR	Compensated	20	1.010	.1518
	Decompensated	20	1.345	.2704

Table 1. Comparison of HA-PI in 2 groups

HAPI	COMPENSATED	DECOMPENSATED	P value
Mean	**1.75**	**1.92**	**< 0.001**
Standard deviation	**0.69**	**0.86**	**< 0.001**

The above table shows comparison of HA-PI between 2 groups. Normal HA-PI is 1.3 to 1.5. HA-PI was found to be high than normal in both groups indicating increased resistance due to cirrhosis, but among the 2 groups it was found to be higher in decompensated group (1.92).

Table 2.Comparison of HA-PI with CTP score

HAPI	CTP A	CTP B	CTP C	P value
Mean	**1.75**	**1.87**	**1.96**	**< 0.001**
Standard deviation	**.070**	**.057**	**0.200**	

In the above table HA-PI correlated with CTP score. It is high in those with CTP C indicating increased severity of liver disease. It had significant correlation with p value <0.001.

Table.3 Comparison of HA-RI in 2 groups

HARI	COMPENSATED	DECOMPENSATED	P value
Mean	**0.69**	**0.87**	**< 0.001**
Standard deviation	**0.046**	**0.055**	

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The table 3 shows HA-RI higher in decompensated group (0.87). It is an indicator of progression of severity of liver disease. It also predicts decompensation of liver disease.

Table 4. Showing Correlation of UGI bleed with HARI and HAPI

	UGI bleed	N	Mean	P value
HA RI	**Present**	**29**	**.8236**	**<0.05**
	Absent	**11**	**.7216**	
HA PI	**Present**	**29**	**1.9173**	
	Absent	**11**	**1.8110**	

Pts with UGI bleed with high HAPI and HARI had a significant p value of < 0.05.

	Encephalopathy	N	Mean	Std. Deviation	P value
HA RI	**Present**	**9**	**.8733**	**.04472**	**0.039**
	Absent	**31**	**.7513**	**.09612**	
HA PI	**Present**	**9**	**1.9233**	**.18661**	**0.361**
	Absent	**31**	**1.8161**	**.11941**	

Correlation of encephalopathy with HAPI and HARI had positive correlation but had no significant correlation.

Table 5. Comparison of HA-RI with CTP scores

HA-RI	CTP A	CTP B	CTP C
MEAN	**0.69**	**0.86**	**0.86**
STANDARD DEVIATION	**0.046**	**0.044**	**0.066**

The table shows comparison of CTP with HA-RI. The HA-RI increased in CTP B and C indicating increased progression of disease.

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Table 6. Showing Comparison of MELD in 2 groups

			Group		Total	P value
			Compensated	Decompensated		
Meld	<= 10	Count	18	0	18	< 0.001
		% within Meld	100.0%	.0%	100.0%	
	> 10	Count	2	20	22	
		% within Meld	9.1%	90.9%	100.0%	
Total		Count	20	20	40	
		% within Meld	50.0%	50.0%	100.0%	

MELD < 10 seen in 20cases and > 10 seen in 20 cases. MELD is indicator of severity of liver disease. We found a significant correlation with MELD in 2 groups with p value < 0.001.

Table 7. Showing Mean and Standard Deviation of HARI and HAPI between two groups

	Group	N	Mean	Std. Deviation
HA RI	**Compensated**	**20**	**.6925**	**0.044**
	Decompensated	**20**	**.8650**	**.05511**
HA PI	**Compensated**	**20**	**1.7590**	**.07055**
	Decompensated	**20**	**1.9215**	**.15006**
Meld	**Compensated**	**20**	**9.50**	**.946**
	Decompensated	**20**	**13.80**	**2.016**

The following table shows significant association of HA-PI, HA-RI and MELD with p value of < 0.001.

Table 8. Showing Comparison of Variable/HAPI/HARI/MELD

Variable	COMPENSATED	DECOMPENSATED	P value
HAPI	**1.76**	**1.92**	**< 0.001**
HARI	**0.69**	**0.87**	**< 0.001**
MELD	**9.5**	**13.8**	**< 0.001**

HAPI and HARI higher in decompensated group as shown in above table. MELD is a valuable indicator of severe liver disease. HAPI is an indicator of resistance indicating PHT. HARI indicates progression to decompensated liver disease. It may be used as a noninvasive marker of progression of liver disease.

All the statistical analysis was carried using the SPSS 16 software. Pearson's correlation coefficient and ANOVA used for analysis of the variables. Paired't' test was used.

DISCUSSION

Doppler parameters such as Hepatic artery Pulsatile index (HAPI) and Resistive index (HARI) were evaluated by **Schneider et al, El Kabanny et al** and studied the correlation between HARI, HAPI and clinical features and complications pertaining to liver disease. Hepatic artery Resistive index (HARI) has also been studied to assess the tendency of liver disease to decompensate.

Liver cirrhosis is characterized by changes in portal and splanchnic circulation[32- 38] as reported in their studies by **Ohnishi et al, Sacerdoti et al** Splanchnic vasodilation is associated with increased resistance to portal venous blood flow. Doppler ultrasonography has been a noninvasive method for evaluation of portal and renal hemodynamics. Portal vein flow has been found low in cirrhotics. In cirrhotics hepatic artery contributes to majority of blood flow as the portal venous flow is reduced in cirrhotics as reported by **Schneider et al [43]** in his studies.

Monitoring of hepatic artery indices such as Hepatic artery Pulsatile index and Resistive index predicts progression to decompensation and severe form of liver disease. Hepatic artery Pulsatile index is considered a better index of resistance and increased portal pressure.

Hepatic arterial resistive indices (HARI) have been shown to increase in cirrhosis and portal hypertension. In this study it was found HAPI to be higher in decompensated (1.92) compared to compensated group (1.76).Higher the HAPI more is the resistance and portal pressure.

Similar findings has been reported by **Schneider et al [43]** who found that Hepatic arterial Pulsatile index (HAPI) to be higher (1.96) in cirrhotics compared to controls in his study and found to have direct correlation with portal pressure. Hepatic arterial pulsatility index (HAPI) correlated with non-invasive assessment of portal hypertension and it has been studied and considered as an equivalent of HVPG.

In this study it was found Hepatic Resistive index to be higher in decompensated group (0.87) compared to compensated group (0.69) with statistical significance (p value < 0.001).Similar association has been reported by **Rivolta et al [5]** who found in his studies that higher the Hepatic RI (0.88) in cirrhotics, the more severe is the ascites and more is the severity of liver disease.

Coli et al [21] also reported in his studies HAPI and HARI can predict severity of fibrosis and liver disease with a specificity of 0.95 and sensitivity of 0.54. He also reported in his study Doppler sonography as safe and noninvasive

method for assessment of HAPI and HARI and correlates well with portal pressure. Most important disadvantage is it is highly operator dependent.

In this study a total of 40 patients of chronic liver disease were enrolled. There were 32 males and 8 females. Etiology of chronic liver disease was evaluated. Alcohol was found to be the commonest cause, followed by Hepatitis B viral infection and Autoimmune hepatitis.

The average hemoglobin was 8.5gm in decompensated group. Low hemoglobin in decompensated group was due to increased grade of varices, increased tendency for variceal bleed, anemia of chronic liver disease, poor oral intake, and bone marrow suppression. The platelet count was 58,700 in decompensated group. It may be due to PHT and hypersplenism. The AST in compensated was 63 IU and 159 IU in decompensated group. In this study we correlated AST which is a marker of fibrosis with Hepatic arterial Pulsatile index(HAPI) and found significant correlation (p value < 0.05).Similar studies[42] done by **[Imber et al]** have shown elevated AST, low platelet count as individual marker of fibrosis and cirrhosis. This forms the basis for use of these variables in yet another valuable score **"APRI"** score.

In this study it was found that HAPI (1.92) and HARI (0.87) to be higher in decompensated group compared to compensated group. Similar association has been reported by **El Kabanny ZA et al [44]** in his studies who also found HAPI (1.98) and HARI (0.90) to be higher in decompensated patients especially Child C group.

In this study it was found that Hepatic artery Resistive index to be higher in decompensated group (0.87) compared to compensated group (0.69) and it was statistically highly significant with p value of < 0.001. We also found positive correlation between HA-PI and AST which was found to be statistically significant (p value <0.05). Above finding indicates the more severe is fibrosis, more is severity of liver disease seen in decompensated group.

Liver biopsy was carried after evaluation of coagulation profile .Since the study population selected for histological correlation was compensated cirrhosis, all patients had F4 fibrosis using Metavir fibrosis score we were unable to carry out histological correlation of these doppler parameters.

We also tried to correlate between CTP score, MELD with HAPI and HARI. MELD was found higher in decompensated group.In our study we found higher the MELD, higher is the HAPI (1.92) and HARI (0.87). **Patrick Kamath et al** [45] also reported similar findings in his studies labeling MELD as predictor of severity of liver disease.

Using Chi square test statistical analysis carried out between variables such as HAPI, HARI and MELD found to have significant p value of < 0.001. We also tried to correlate platelet count with HAPI and HARI, we had positive correlation but found no significant correlation between them.

In this study we found HAPI (1.92) and HARI (0.87) to be higher in decompensated group with statistical significance (p value <0.001) indicating predictors of severity of liver disease. Moreover by monitoring of Hepatic artery Resistive index (HARI) by serial Doppler assessment we can predict which group of patients have tendency to decompensate, initiating aggressive management of portal hypertension.

CONCLUSION

1. HAPI is indicator of severity of resistance due to fibrosis in both study groups.
2. HAPI and HARI similar to that of MELD are non invasive doppler parameters predicting severity of liver disease and its progression.
3. Hepatic artery pulsatile index had significant correlation with AST, indicating severe fibrosis and severe form of liver disease.
4. All patients in compensated group had cirrhosis (F4) hence histological correlation could not be carried out.

We need further large studies to validate the outcome of this study.

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ABBREVIATIONS

PHT- Portal Hypertension

HA-RI - Hepatic artery Resistive index

HA-PI -Hepatic artery Pulsatile index

CTP – Child Turcotte Pugh score

ECM- Extracellular Matrix

ICS-Intercostal space

HSC- Hepatic Stellate cells

PDGF- Platelet derived growth factor

TGF-β – Transforming growth factor

EGF- Epidermal growth factor

IGF- Insulin like growth factor

HVPG- Hepatic vein pressure gradient

SMA- Superior mesenteric artery

AST- Aspartate aminotransferases

USG- Ultrasound

PV- Portal vein

OGD- Esophagogastroduodenoscopy

PSV- Peak systolic velocity

PDV- Peak diastolic velocity

Assessment of Hepatic Doppler parameters in Cirrhotics its clinical and Histological correlation - Proforma

Name		IP no		D.O.A	
Age	+	Unit		D.O.Discharge D.O.Death	
Sex		Ward		Duration of stay	
Address			Diagnosis		
Phone No.					
History					
Jaundice			Altered sensorium		
Abdominal Distension			Hematemesis		
Pedal edema			Malena		
Oliguria			Weight loss		
Puffiness of face			Spontaneous bleeding		
Fever			Muscle cramps		
Anorexia			Cough		
fatigue			breathlessness		
Constipation					
Diarrhea					
Native medication					
Past h/o jaundice			Tattooing		
Diabetes			Blood transfusion		
Smoking			Drug abuse		
Alcohol Duration Gm/day					

Examination					
HE grade		Clubbing		PR	
Nutrition		Cyanosis		RR	
Height		Parotid swelling		Temp	
Weight		Gynaecomastia		BP Systolic	
BMI		Palmar erythema		Diastolic	
Anaemia		Scrotal swelling		Pulse pressure	
Icterus		Skin changes		Neck veins	
Pedal edema		Abd veins		CVS	
Ascites		Back veins		RS	
Umblical hernia		Caput medusae			
Splenomegaly		Hepatomegaly			
Investigation					
USG Abdomen Liver Size Echoes Ascites Spleen			Endoscopy		
PV Doppler			CXR		
Ascitic fluid culture Colour SAAG Cell count					
CTP			Urine culture		

Date					
TC					
Hb					
Platelet					
RBS					
Urea					

Creatinine					
Sodium					
Potassium					
T.Bilirubin					
Direct					
Indirect					
SGOT					
SGPT					
ALP					
Protein					
Albumin					
globulin					
PT					
INR					

LIVER BIOPSY

GRADE OF FIBROSIS
F0
F1
F2
F3
F4

LIVER PARAMETERS

portal venous time-averaged maximum velocity	
HA maximum peak systolic velocity (HA-PSV)	
HA Peak diastolic velocity (HA-PDV)	
Mean velocity	

Hepatic artery Pulsatile index= $\frac{\text{Peak systolic velocity- Peak diastolic velocity}}{\text{Mean velocity}}$

Hepatic artery Resistive index= $\frac{\text{Peak systolic velocity- Peak diastolic velocity}}{\text{Peak systolic velocity}}$

INFORMATION SHEET

We are conducting a study on Doppler parameters of Hepatic Hemodynamics in cirrhotics and to correlate them with clinical and histological parameters at the Department of Medical Gastroenterology, Rajiv Gandhi Government General Hospital, Chennai. The purpose of study is to compare various Doppler parameters of Hepatic and Renal Hemodynamics and correlate clinically, biochemically and histologically in Compensated Cirrhotics.

The privacy of the patient will be maintained throughout the study .In the event of any publication or presentation resulting from research no personal information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in the study or to withdraw at anytime. Your decision will not result in any loss of benefits to which you are entitled.

The results of the study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the investigator Signature of the participant

INFORMED CONSENT FORM

Title of the Study:

Doppler parameters of Hepatic Hemodynamics in Cirrhotics and its Clinical and Histological correlation

Name of the Participant:

_____

Name of the Investigator: Dr. Karthikeyan .R

Name of the Institution : Madras Medical College.

Documentation of the informed consent

I _____ have read the information of this form (or it had been read to me). I was free to ask any questions and they have been answered. I hereby give my consent to be included as a participant in

Doppler parameters in Hepatic and Renal Hemodynamics in Cirrhotics and its Clinical and Histological correlation.

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.

Name and signature / thump impression of the participant

Name _____ Signature _____ Date____

Name and signature of impartial witness (required for illiterate patients):

Name _____ Signature _____ Date____

Address and contact number of the impartial witness:

Name and signature of the investigator or his representative obtaining consent:

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

"கல்லீரல் நோயினால் பாதிக்கப்பட்டவர்களைப் பற்றிய ஆய்வு"

ஆராய்ச்சி நிலையம் : இராஜீவ்காந்தி அரசு பொது மருத்துவமனை
சென்னை மருத்துவக்கல்லூரி,
சென்னை - 600 003.

பங்கு பெறுபவரின் பெயர் :

பங்குபெறுபவரின் எண் :

பங்கு பெறுவர் இதனை (✓) குறிக்கவும்.

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தகமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். ☐

இந்த ஆய்வில் எனக்கு இரத்தம், சிறுநீர், ஊடுகதிர் படம் மற்றும் மின் உடலியங்கியல் பரிசோதனை செய்துகொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம் ..... இடம் ..... தேதி

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர் .....

நோயாளியின் உறவினர்/காப்பாளர் கையொப்பம் ..... இடம் ..... தேதி

ஆராய்ச்சி தகவல் தாள்

சென்னை இராஜீவ்காந்தி அரசு பொது மருத்துவமனைக்கு வரும் நோயாளிகளில், கல்லீரல் நோயினால் பாதிக்கப்பட்டவர்கள் குறித்த ஆய்வு இங்கு நடைபெற்று வருகிறது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க விரும்புகிறோம். இந்த ஆராய்ச்சியில் உங்களை பங்கேற்க வைத்து அதன் தகவல்களை ஆராய்வோம். அதனால், தங்களின் நோயின் ஆய்வறிக்கையோ, சிகிச்சையோ பாதிக்கப்படாது என்பதைத் தெரிவித்துக் கொள்கிறோம்.

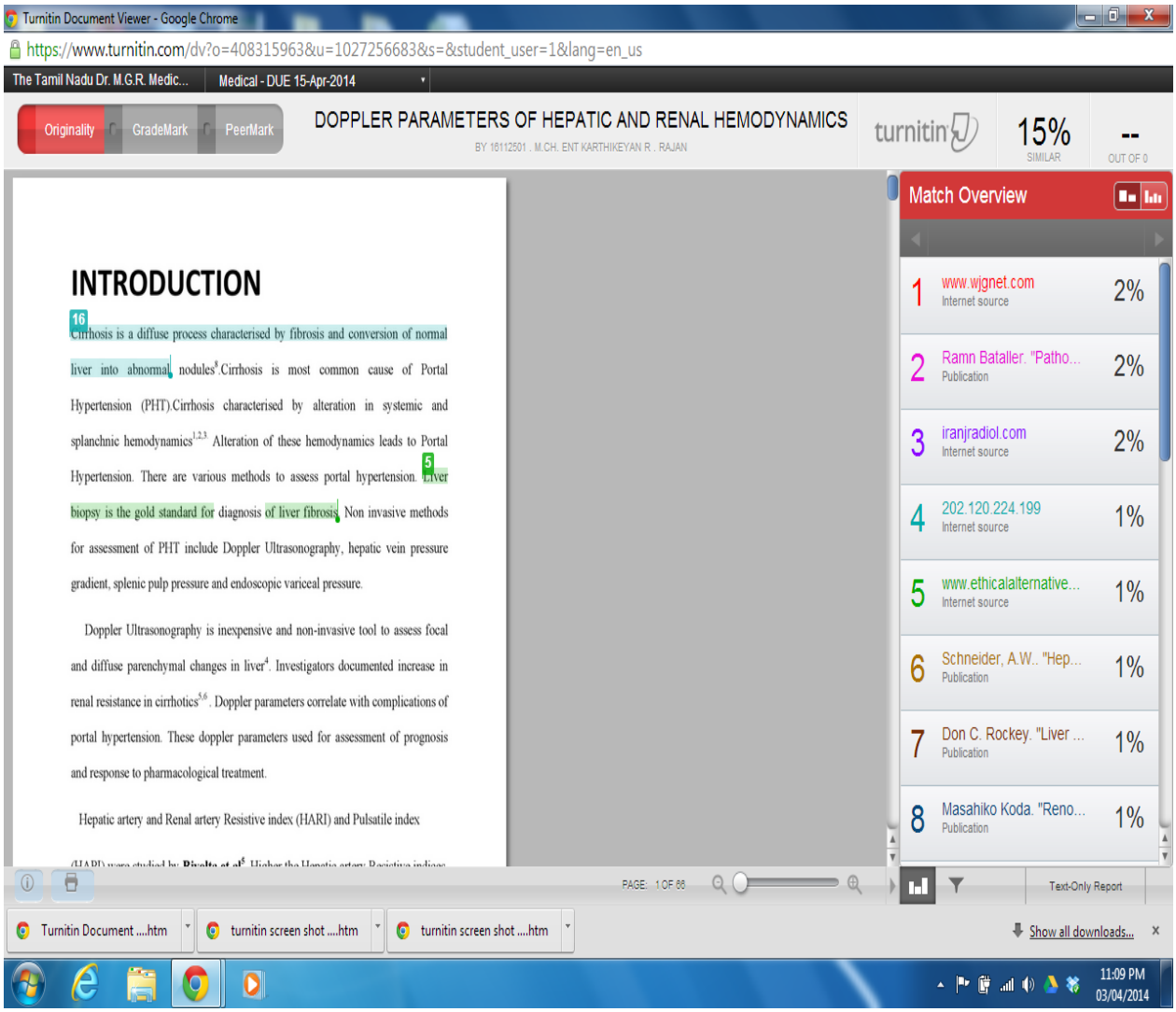
முடிவுகளை அல்லது கருத்துகளை வெளியிடும்போதோ அல்லது ஆராய்ச்சின்போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின் வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம்

பங்கேற்பாளர் கையொப்பம்

பங்கேற்பாளரின் உறவினர் கையொப்பம்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INTRODUCTION
Cirrhosis is a diffuse process characterised by fibrosis and conversion of normal liver into abnormal nodules[8].Cirrhosis is most common cause of Portal Hypertension (PHT).Cirrhosis characterised by alteration in systemic and splanchnic hemodynamics[1,2,3]. Alteration of these hemodynamics leads to Portal Hypertension. There are various methods to assess portal hypertension. Liver biopsy is the gold standard for diagnosis of liver fibrosis. Non invasive methods for assessment of PHT include Doppler Ultrasonography, hepatic vein pressure gradient, splenic pulp pressure and endoscopic variceal pressure.
Doppler Ultrasonography is inexpensive and non-invasive tool to assess focal and diffuse parenchymal changes in liver[4]. Investigators documented increase in renal resistance in cirrhotics[5,6]. Doppler parameters correlate with complications of portal hypertension. These doppler parameters used for assessment of prognosis and response to pharmacological treatment.
Hepatic artery and Renal artery Resistive index (HARI) and Pulsatile index
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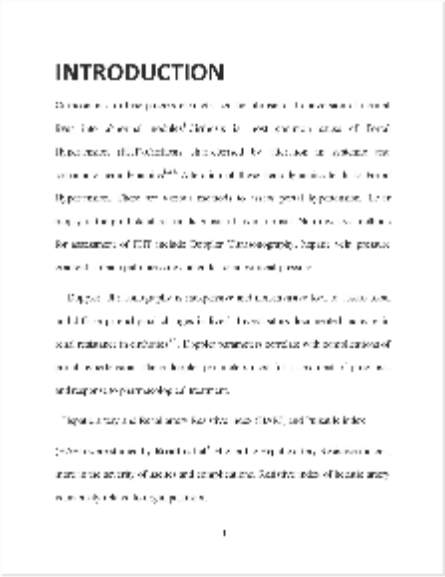

INTRODUCTION

Group	name	Age in years	Sex	GE No	jaundice	ascites	UGI bleed	Encephalop	alcohol	smoking	diabetes	Hepatome	Splenomeg	wbc	hb
Compensated	SANTHANAM	45	Male	6405/13	Absent	Absent	Present	Absent	Present	Present	Absent	Absent	Present	5400	14
Compensated	RAJU	45	Male	196/14	Absent	Absent	Present	Absent	Present	Present	Absent	Present	Present	6700	13
Compensated	VENKATESAN	50	Male	78/14	Absent	Absent	Present	Absent	Present	Absent	Absent	Present	Present	6400	10
Compensated	PALANI	59	Male	6177/13	Absent	Absent	Absent	Absent	Present	Present	Absent	Absent	Present	4700	12
Compensated	MUTHU	37	Male	156/14	Absent	Absent	Present	Absent	Present	Present	Absent	Absent	Present	6400	10
Compensated	RAMU	44	Male	186/14	Absent	Absent	Present	Absent	Present	Absent	Absent	Present	Present	5500	12
Compensated	THENARASU	48	Male	6133/13	Absent	Absent	Absent	Absent	Present	Present	Absent	Present	Absent	5400	10
Compensated	RAJENDIRAN	50	Male	5455/13	Absent	Absent	Present	Absent	Present	Present	Absent	Absent	Absent	4400	10
Compensated	POTHU	35	Female	5571/13	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	Present	4500	9
Compensated	SURESH	37	Male	6033/13	Absent	Absent	Present	Absent	Present	Present	Absent	Present	Absent	7700	9.5
Compensated	NEELA	38	Female	6700/13	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present	6800	10
Compensated	JEYARAJ	40	Male	4988/13	Absent	Absent	Present	Absent	Present	Absent	Absent	Absent	Present	4500	14
Compensated	RAJALAKSHMI	43	Female	5933/13	Absent	Absent	Present	Absent	Absent	Absent	Absent	Absent	Present	6500	9.8
Compensated	ANBU	48	Male	5680/13	Absent	Absent	Present	Absent	Present	Present	Absent	Present	Present	7000	11.2
Compensated	NAGARAJ	49	Male	5589/13	Absent	Absent	Present	Absent	Present	Present	Absent	Absent	Present	4700	14
Compensated	VEDACHALAM	50	Male	5809/13	Absent	Absent	Present	Absent	Present	Absent	Absent	Absent	Present	6800	9.6
Compensated	GOVINDAN	55	Male	5890/13	Absent	Absent	Present	Absent	Present	Absent	Absent	Present	Present	5500	9
Compensated	ADHILAKSHMI	60	Female	68/14	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	Present	7000	13
Compensated	VIJAYAKUMAR	38	Male	5785/13	Absent	Absent	Present	Absent	Present	Present	Absent	Absent	Present	9500	12
Compensated	VASANTHA	40	Female	6138/13	Absent	Absent	Present	Absent	Absent	Absent	Absent	Present	Present	5500	9.2
Decompensated	DAMODARAN	65	Male	34/14	Present	Present	Present	Absent	Present	Present	Absent	Present	Present	6500	9
Decompensated	KUPPUSAMY	34	Male	6070/13	Present	Present	Absent	Present	Present	Present	Absent	Absent	Present	5600	6.8
Decompensated	DASS	35	Male	5568/13	Present	Present	Present	Present	Present	Absent	Absent	Present	Absent	6800	10
Decompensated	VALIAMMAL	42	Female	5509/13	Present	Present	Present	Present	Absent	Absent	Absent	Absent	Present	7000	8.7
Decompensated	ARUMUGAM	60	Male	108/14	Present	Present	Present	Absent	Present	Present	Absent	Present	Present	5400	9
Decompensated	KIRUBAKARAN	63	Male	5750/13	Present	Present	Present	Absent	Present	Present	Absent	Absent	Present	5300	7
Decompensated	RAJKUMAR	42	Male	4783/13	Present	Present	Present	Absent	Present	Present	Absent	Absent	Present	4300	9
Decompensated	PITCHAIKARAN	42	Male	6279/13	Present	Present	Absent	Absent	Present	Present	Absent	Absent	Present	6500	7.8
Decompensated	VENKATAMMAL	45	Female	6144/13	Present	Present	Present	Absent	Absent	Absent	Absent	Absent	Present	6600	9.4
Decompensated	MUTHU	33	Male	6074/13	Present	Present	Present	Absent	Present	Absent	Absent	Absent	Present	7000	7.7
Decompensated	Rangachari	55	Male	128/14	Present	Present	Absent	Present	Absent	Absent	Absent	Present	Present	7500	8.3
Decompensated	Veeraragavan	57	Male	165/14	Present	Present	Present	Absent	Present	Present	Absent	Absent	Present	4800	7.2
Decompensated	Vasanthakumar	24	Male	5919/13	Present	Present	Present	Absent	Absent	Absent	Absent	Absent	Present	4600	9.2
Decompensated	Raju	60	Male	1638/14	Present	Present	Absent	Present	Present	Present	Absent	Absent	Present	5900	9.2
Decompensated	Kaniyappan	69	Male	1620/14	Present	Present	Absent	Absent	Present	Present	Absent	Absent	Absent	4200	8.4
Decompensated	Abdul kaboor	56	Male	1217/14	Present	Present	Present	Present	Present	Absent	Absent	Present	Present	10900	9.2
Decompensated	Neelakandan	35	Male	1635/14	Present	Present	Present	Absent	Present	Absent	Absent	Absent	Present	6900	10
Decompensated	Jeyakrishnan	48	Male	4657/11	Present	Present	Present	Present	Present	Absent	Absent	Absent	Absent	4400	7.4
Decompensated	Raman	56	Male	1436/14	Present	Present	Absent	Present	Present	Present	Absent	Absent	Present	5600	9.6
Decompensated	saroja	48	Female	486/14	Present	Present	Absent	Present	Absent	Absent	Absent	Absent	Present	8600	8.4

Group	name	Age in years	Sex	proteins	albumin	ast	alt	BILIRUBIN	pt	inr	varices	HA RI	HA PI	etiology	meld	meld_g	CTP
Compensated	SANTHANAM	45	Male	6	3.9	68	49	1	17	0.9	Grade I	0.70	1.70	Alcohol	8	<= 10	A
Compensated	RAJU	45	Male	7	3.5	70	43	0.9	16	1	Grade II	0.70	1.87	Alcohol	8	<= 10	A
Compensated	VENKATESAN	50	Male	6	3.8	68	36	1	19	1.1	Grade1	0.75	1.82	Alcohol	8	<= 10	A
Compensated	PALANI	59	Male	7	3.8	57	40	1	18	1.2	No Varices	0.73	1.70	HBV	8	<= 10	A
Compensated	MUTHU	37	Male	6	3.6	64	40	1.1	16	0.8	Grade I	0.74	1.80	Alcohol	9	<= 10	A
Compensated	RAMU	44	Male	7	3.8	76	46	1	17	0.9	no varices	0.76	1.70	Alcohol	9	<= 10	A
Compensated	THENARASU	48	Male	6	2.9	64	46	2	17	0.8	Grade I	0.70	1.80	Alcohol	9	<= 10	A
Compensated	RAJENDIRAN	50	Male	7	3.6	56	39	0.9	18	0.8	Grade I	0.68	1.76	HBV	9	<= 10	A
Compensated	POTHU	35	Female	7	3.5	48	45	0.8	19	1.2	GradeI	0.74	1.70	Alcohol	10	<= 10	A
Compensated	SURESH	37	Male	7	4	63	47	0.9	19	1.2	GradeI	0.65	1.80	Alcohol	10	<= 10	A
Compensated	NEELA	38	Female	6	4	90	64	1	17	1	GradeI	0.72	1.80	HBV	10	<= 10	A
Compensated	JEYARAJ	40	Male	6	3.7	66	47	1	16	0.9	GradeI	0.62	1.64	Autoimmu	10	<= 10	A
Compensated	RAJALAKSHMI	43	Female	7	4.3	72	54	1	16	0.8	GradeI	0.60	1.86	HBV	10	<= 10	A
Compensated	ANBU	48	Male	7	4.4	26	36	1	17	1.1	No Varices	0.64	1.60	Alcohol	10	<= 10	A
Compensated	NAGARAJ	49	Male	6	3	54	44	1	17	1.2	GradeI	0.73	1.78	Alcohol	10	<= 10	A
Compensated	VEDACHALAM	50	Male	6	3.4	70	45	0.9	17	1.1	Grade I	0.62	1.76	Alcohol	10	<= 10	A
Compensated	GOVINDAN	55	Male	7	4	64	46	1.1	18	0.9	Grade I	0.72	1.70	Alcohol	10	<= 10	A
Compensated	ADHILAKSHMI	60	Female	6	3	66	40	0.85	19	1.1	Grade I	0.70	1.80	AUTOIMMI	10	<= 10	A
Compensated	VIJAYAKUMAR	38	Male	6	3.6	55	49	0.9	16	1.2	GradeI	0.66	1.79	Alcohol	11	> 10	A
Compensated	VASANTHA	40	Female	7	3.5	76	56	3	19	1	Grade II	0.69	1.80	HBV	11	> 10	A
Decompensated	DAMODARAN	65	Male	7	3	160	120	6.5	18	1.2	Grade I	0.80	1.90	Alcohol	11	> 10	B
Decompensated	KUPPUSAMY	34	Male	6	3.4	170	105	7	19	1.1	No Varices	0.85	1.90	Alcohol	12	> 10	B
Decompensated	DASS	35	Male	7	3	220	160	12	20	1.4	Grade II	0.90	1.85	Alcohol	13	> 10	C
Decompensated	VALIAMMAL	42	Female	6	3.5	165	135	9	18	1.1	Grade II	0.85	1.80	HBV	12	> 10	B
Decompensated	ARUMUGAM	60	Male	6	3.7	220	145	7	17	0.8	Grade II	0.80	1.87	Alcohol	12	> 10	B
Decompensated	KIRUBAKARAN	63	Male	7	4	260	150	5	18	1.3	Grade III	0.75	1.80	HBV	14	> 10	C
Decompensated	RAJKUMAR	42	Male	6	3.6	140	110	4	19.5	1.4	Grade III	0.94	1.85	Alcohol	13	> 10	B
Decompensated	PITCHAIKARAN	42	Male	7	3.4	76	45	5.6	18.6	1.3	GradeII	0.86	1.95	HBV	14	> 10	B
Decompensated	VENKATAMMAL	45	Female	7	3.6	110	86	6	23	1.6	Grade II	0.90	1.90	HBV	15	> 10	C
Decompensated	MUTHU	33	Male	6	3.5	86	65	7.6	22	1.5	GradeIII	0.86	1.86	Autoimmu	15	> 10	C
Decompensated	Rangachari	55	Male	6	3	176	134	8.7	19	1	Grade II	0.85	1.90	cryptoge	11	> 10	B
Decompensated	Veeraragavan	57	Male	6	2.9	156	106	9	27	2	Grade III	0.82	1.84	alcohol	19	> 10	C
Decompensated	Vasanthakumar	24	Male	6	3.4	130	97	7	18	1.1	Grade III	0.85	1.85	alcohol	12	>10	C
Decompensated	Raju	60	Male	6	2.9	156	90	6	21	1.3	Grade II	0.95	2.40	alcohol	14	> 10	C
Decompensated	Kaniyappan	69	Male	6	3	106	88	8	18.7	1.4	Grade II	0.90	2.20	alcohol	14	<= 10	C
Decompensated	Abdul kaboor	56	Male	6	3.6	92	79	7	21	1.4	GradeII	0.80	1.85	HBV	16	> 10	C
Decompensated	Neelakandan	35	Male	6	2.8	170	105	8	20	1.4	GradeII	0.96	2.10	alcohol	14	> 10	C
Decompensated	Jeyakrishnan	48	Male	6	3.4	200	140	6.5	18	1.5	Grade III	0.86	1.97	alcohol	15	> 10	B
Decompensated	Raman	56	Male	6	3	210	137	6	18	1.3	GradeII	0.92	1.80	alcohol	13	> 10	B
Decompensated	saroja	48	Female	6	2.8	180	110	7	18	1.8	Grade III	0.88	1.84	HBV	17	> 10	B